JAN 18 1923 Medical Lib.

Vol. V JANUARY, 1923 No. 1

THE AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY

ADVISORY EDITORIAL BOARD

CHANNING W. BARRETT
C. L. BONIFIELD
J. WESLEY BOVEE
W. W. CHIPMAN
JOHN G. CLARK
H. S. CROSSEN
THOMAS CULLEN
EDWARD P. DAVIS
J. B. DELEE
ROBERT L. DICKINSON
PALMER FINDLEY
ROBERT T. FRANK
GEORGE GELLHORN
ALBERT GOLDSPOHN
WILLIAM P. GRAVES
HERMAN E. HAYD
BARTON C. HIRST
E. J. ILL
J. C. LITZENBERG
F. W. LYNCH
FRANKLIN H. MARTIN
C. JEFF MILLER
GEORGE CLARK MOSHER
HENRY P. NEWMAN
GEO. H. NOBLE
REUBEN PETERSON
JOHN OSBORN POLAK
F. F. SIMPSON
HENRY SCHWARZ
J. M. SLEMONS
HOWARD C. TAYLOR
THOMAS J. WATKINS
B. P. WATSON
GEORGE GRAY WARD, JR.
J. WHITRIDGE WILLIAMS

OFFICIAL ORGAN OF
THE AMERICAN GYNECOLOGICAL SOCIETY
THE AMERICAN ASSOCIATION OF OBSTETRICIANS, GYNECOLOGISTS, AND ABDOMINAL SURGEONS
NEW YORK OBSTETRICAL SOCIETY; OBSTETRICAL SOCIETY OF PHILADELPHIA; BROOKLYN GYNECOLOGICAL SOCIETY; AND ST. LOUIS GYNECOLOGICAL SOCIETY

Editor, GEORGE W. KOSMAK
Associate Editor, HUGO EHRENFEST

Published Monthly by
THE C. V. MOSBY COMPANY
ST. LOUIS

—Foreign Depots—

HIRSHFELD BROS., Ltd., London, England.
STIRLING & COMPANY, Melbourne, Australia.
EMILE BOUGAULT, Paris, France.
EDWARD EVANS & SONS, Ltd., Shanghai, China.
McAINSH & COMPANY, Ltd., Toronto, Canada.
MARUZEN COMPANY, Ltd., Tokyo, Japan.

(Entered as Second-Class Matter October 28, 1920, at the Post Office at St. Louis, Mo., under the Act of March 3, 1879).

Each new year
brings to
D & G Sutures
New Excellence
and New Friends

See the insert pages in this issue
for full information as to products and prices

DAVIS & GECK, INC.
Surgical Sutures Exclusively
211 to 221 Duffield Street - Brooklyn, N.Y., U.S.A.

COPYRIGHT JAN 1923 D&G INC

for GYNECOLOGISTS
and OBSTETRICIANS

Neet

Sterile, antiseptic, non-irritant

A bland alkaline cream that removes hair without irritating the skin.

Relieves patient of the discomfort and risk attending the use of a razor.

Incoming growth is slower to reach the surface than if shaved, and is softer and finer than growth following use of a razor.

A liberal trial sample with complete instructions for use will be mailed without charge to any physician requesting it.

HANNIBAL PHARMACAL COMPANY—St. Louis, Mo.

med.
library

The American Journal of Obstetrics and Gynecology

VOL. V. ST. LOUIS, JANUARY, 1923 NO. 1

Original Communications

THE MICROSCOPIC AS COMPARED WITH THE CLINICAL DIAGNOSIS OF MALIGNANT UTERINE NEOPLASM

BY CHARLES C. NORRIS, M.D., PHILADELPHIA, PA.

From the Laboratory of Gynecologic Pathology, University of Pennsylvania

MOST investigators recognize the value of making a routine histologic examination of all neoplastic tissue removed at operation. This study has been undertaken with a view to checking up with the actual figures the relative values of this procedure among the different types of malignant tumors.

TABLE I

CARCINOMA OF THE CERVIX

Total number of cases	253	
Squamous cell carcinoma	219	
Adenocarcinoma	34	
Early stage	41	(16%)
Moderately advanced stage	103	(40%)
Advanced stage	109	(43%)
Clinical diagnosis positive	206	(81.4%)
Clinical diagnosis carcinoma cervicitis? (doubtful diagnosis)	29	(11.4%)
Clinical diagnosis benign, but microscopic examination showed malignancy	6	(2.3%)
Clinical diagnosis malignancy, but microscopic examination showed benign	3	(1.5%)
Clinical diagnosis carcinoma of fundus, microscopic examination showed cervical origin	10	(3.9%)
Clinical diagnosis sarcoma of the cervix, microscopic examination showed carcinoma	2	(0.7%)

CARCINOMA OF THE CERVIX

Cancer of the cervix can generally be diagnosed correctly by clinical methods. In our series of 253 specimens, 81.4 per cent were diag-

NOTE: The Editor accepts no responsibility for the views and statements of authors as published in their "Original Communications."

nosed positively by the clinician, 11.4 per cent were suspected. In only 2.3 per cent was malignancy unsuspected by the clinicians.

In carcinoma of the cervix the lesion is not only fairly characteristic clinically, but is easily accessible to sight and touch, and can be examined thoroughly during the course of an ordinary office examination. Furthermore, in doubtful cases, biopsy is a simple procedure. In considering the high percentage of correct clinical diagnoses it should be remembered that these cases were studied by specialists, and the proportion of correct clinical diagnoses is, therefore, probably considerably higher than it would have been if the same cases had been observed in general practice.

There are a few conditions, however, which so closely simulate carcinoma that the correct diagnosis is almost impossible from clinical signs alone. I have seen two cases of tuberculosis of the cervix, in one of which the correct diagnosis was suspected, but in the other case carcinoma was diagnosed positively. Tuberculosis of the cervix generally produces a lesion that closely resembles carcinoma, and because of the rarity of the former condition, a diagnosis of carcinoma is usually made. In a series of 128 cases of tuberculosis of the cervix collected from the literature (Norris, C. C.: Gynecologic and Obstetric Tuberculosis, D. Appleton and Co., N. Y., 1921), over 88 per cent were diagnosed clinically as malignant. Syphilis may also produce a lesion that will strongly suggest carcinoma. Extensive laceration with eversions, particularly when complicated by pregnancy, is often suggestive of malignancy. Adenomyoma of the rectovaginal septum (Cullen) may produce a condition that closely simulates carcinoma, but that can easily be differentiated from the latter by microscopic examination.

The percentage of operability in cases reported of carcinoma of the cervix varies in different clinics. Graves' (Surgery, Gynecology, and Obstetrics, June, 1921) latest figures show an operability of 64 per cent. Of the 253 cases studied, but 16 per cent have been classed as in the early stages. Carcinoma of the cervix gives a relatively small proportion of five year cures, regardless of the method of treatment. The urgent necessity for early diagnosis is, therefore, apparent. Skeel (loc. cit.) states that the expectation of an increased number of cures of cancer of the cervix by surgical methods must be based upon earlier diagnosis, and that panhysterectomy should be reserved for cases in which a positive diagnosis can be made only with the microscope. This statement represents an extreme view, and if it had been applied to the series herein reported, would have reduced the operability to considerably less than 10 per cent. It is not usual, even today, for cases of inoperable carcinoma that have been treated locally for prolonged periods to be referred to surgeons. When the malig-

nant growths are deep seated, the most experienced surgeon may sometimes be in doubt. With the cervical lesions, however, biopsy is so simple an operation and so certain a means of clearing up the doubtful cases that to neglect its use is inexcusable.

CARCINOMA OF THE BODY OF THE UTERUS

In carcinoma of the fundus a different state of affairs exists. Here we have a lesion that can be neither inspected nor palpated, and even the experienced gynecologist is forced to depend more or less, for a final diagnosis, upon the microscopic examination. The method suggested by John G. Clark, which consists of cleansing the vagina and cervix and then passing a sterile sound to the fundus of the uterus, slightly manipulating the instrument in order to determine whether or not bleeding occurs, and also to measure the depth of the uterus, is of distinct value, and is a procedure that may be carried out in the physician's office. Ewing has supplemented this test by taking smears from the fundus, and by this means has been able to demonstrate in a proportion of cases the presence of carcinoma cells or of fragments of the tumor. Whereas these tests are of distinct practical value, they are not conclusive in all cases, particularly in the early ones. Even when the curet withdraws large quantities of brain-like tissue, the diagnosis is not certain until a histologic examination has been made. Such a case recently came under my care. The history and clinical findings were those of carcinoma, and the diagnosis was further strengthened by the macroscopic appearance of the curettings. Histologic examination, however, showed the condition to be a hypertrophic polypoid lesion, and saved the patient the risks and discomforts of a hysterectomy. A cure was effected with radium, and the patient has remained well to date. Such cases are, of course, the exception, but they nevertheless prove the value of routine histologic examination.

There are many intrauterine lesions that may be confused with carcinoma. One of the most common of these is carcinoma combined with myoma. An analysis of the cases studied shows that at least 75 per cent of the mistaken diagnoses were due to this condition. The clinical symptoms of myoma may completely mask those of carcinoma, and for this reason practically all gynecologists make it a rule to perform a curettage prior to employing radium in the treatment of these tumors. Many instances could be cited in which the long-continued menorrhagia of myomata has completely overshadowed the metrorrhagic type of bleeding due to carcinoma. Of the 101 cases of carcinoma of the fundus that were studied no fewer than 20 were clinically diagnosed as benign, 57 were positively diagnosed by clinical means, and an additional 24 were suspected of being malignant. Thus about 20 per cent were of the unsuspected variety. This in itself is suffi-

cient evidence to prove the necessity for making routine histologic examinations. Even after removal of the uterus cases will occasionally be encountered that will prove puzzling until they are subjected to microscopic examination. I have recently seen a specimen of a diffuse, partially submucous adenomyoma undergoing necrosis that left no doubt in my mind as to its malignancy until it was subjected to microscopic examination.

TABLE II

CARCINOMA OF THE BODY OF THE UTERUS

Total number of cases	101	percentage
Early stage	24	23.7
Moderate stage	53	52.4
Advanced stage	24	23.7
Clinical diagnosis positive	57	56.4
Clinical diagnosis doubtful	24	23.7
Clinical diagnosis benign, but proved to be malignant	20	19.8
Clinical diagnosis myoma	15	14.8
Clinical diagnosis metrorrhagia myopathica	2	1.9
Clinical diagnosis polypoid endometritis	2	1.9
Clinical diagnosis unsuspected	1	.9
Clinical diagnosis carcinoma of cervix but proved fundal in origin	1	.9

An analysis of the cases of carcinoma of the fundus that came to my notice, in which curettings were the only specimens submitted, shows the following results. Naturally these comprise the greatest proportion of doubtful cases, for if the diagnosis were positive, curettage would not be indicated. Of the entire series of 101 cases of carcinoma of the fundus, curettings alone were submitted to the laboratory in 58 cases. Of these, the clinical diagnosis was positive in 21 or 36.2 per cent. The clinical diagnosis was doubtful or suspected of malignancy in 22 cases, and in 15, or 25.8 per cent, it was benign. It is impossible to give the figures as regards the frequency with which the clinical diagnosis of malignant tumor was made but which proved to be benign. The latter, however, undoubtedly comprise a considerable group.

For making a successful diagnosis from curettings it is essential that the pathologist possess a thorough knowledge of the changes that take place in the normal endometrium during the menstrual cycle and at different ages, as well as the changes produced by pregnancy. He should, therefore be furnished such data from the history of each case as are necessary. A positive histologic diagnosis is often difficult to arrive at, and the cooperation of the clinician is essential in order to secure the best results. Recently a number of articles have appeared in the gynecologic literature decrying the operation of curettage as a routine procedure. This has no bearing on curettage performed for diagnostic purposes. With the latter end in view, the curettage should be a thorough one, as the carcinomatous lesion

may be small. Obviously it is impossible for the microscopist to make a correct diagnosis unless he receives the tissue. I prefer to have the tissue placed immediately in a bottle of Zenker's solution, and thoroughly shaken in order to avoid the formation of clots. Paraffin sections are the most satisfactory for this work. The paraffin in which the tissue is to be mounted should be hot, in order that the entire tissue may sink as nearly as possible to the same level. Ordinary paper boxes are not suitable for this work, a metal box being preferable. A good plan is to place the tissue in the mounting box, having the paraffin warm, and then to heat the box. This liquefies all the paraffin, and allows the tissue to sink to the bottom in a single layer. In this way all the tissue can easily be cut. Care must, of course, be exercised not to "cook" the tissue. Even with this method it is advisable to take a section, cut down into the block a short distance, take another section until all the tissue has been cut. In this way a portion of every particle of tissue is subjected to microscopic inspection. These suggestions are particularly applicable when there is only a small amount of curettings. The pathologist should be warned in every case in which there is the least suspicion of malignancy, in order that special precautions may be taken.

TABLE III

ADENOCARCINOMA OF THE BODY OF THE UTERUS (CURETTINGS).

Total number of cases	58	
Clinical diagnosis positive	21	(36.2%)
Clinical diagnosis doubtful	22	(37.9%)
Clinical diagnosis benign	15	(25.8%)

SARCOMA

The frequency of occurrence of this type of neoplasm has been greatly overestimated in the past, some pathologists asserting that 10 per cent of all myomata possess malignant characteristics. Sarcoma may originate in two ways: either as a primary malignant tumor, or as a degeneration of a primary benign neoplasm—generally a fibromyoma. In the latter type of tumor some form of degeneration is usually present, and in almost every specimen, if a sufficient number of blocks are examined, an area of markedly cellular appearance will be observed. These latter are without doubt, occasionally mistaken for sarcoma. Our series of sarcoma (including one endothelioma) consists of 35 cases. In contrast to this and removed during the same period of time, there were 1216 fibromyomata of the uterus. Of the 35 malignant connective tissue tumors, only 8 appear to have been degenerations of previously benign neoplasms, showing that in this series sarcomatous degeneration of myomata occurred in only about 0.6 of 1 per cent of cases. In confirmation of the correctness of the microscopic examination in the myomata and sarcomata it may be

stated that follow-ups have been made on both. None of the myomata have had recurrences and over 80 per cent of the sarcomata have succumbed.

The frequency of sarcoma in relation to myoma is of especial interest in view of the increasing use of radium and the x-ray in the treatment of certain forms of the latter. If sarcomatous degeneration were of frequent occurrence, operations would be indicated in practically all cases. Berreitter (Zentralbl. f. Gynäk, 1921, xlv, 1592) in his series of 716 cases of myomata found 6 malignant tumors, and Evans (Surg., Gynec. and Obst., 1920, xxx, 225) examined 4000 myomata from the Mayo Clinic and found 72 cases of sarcoma.

TABLE IV
SARCOMA OF THE UTERUS

Total number	35	percentage
Variety, spindle-celled	11	31.4
Variety, small round cell	7	20
Variety, large round cell	3	8.5
Variety, mixed cell	13	37.1
Endothelioma	1	2.8
Early stage	10	28
Moderately advanced stage	17	48.5
Advanced stage	8	22.8
Clinical diagnosis positive	8	22.8
Clinical diagnosis doubtful	5	14.6
Clinical diagnosis carcinoma but proved sarcoma	3	8.5
Benign but proved malignant	19	54.2
Clinical diagnosis myoma	16	45.7
Clinical diagnosis ovarian cyst	1	2.8
Clinical diagnosis endometritis	1	2.8
Clinical diagnosis pelvic inflammatory disease	1	2.8

1 case of sarcoma of the cervix, 1 case of sarcoma of the cervical stump.

CHORIOEPITHELIOMA

Various classifications of these tumors have been suggested. Chorioepitheliomata vary markedly as regards malignancy. Many efforts have been made to correlate the malignancy of the various types with their histologic appearance, but these have met with only limited success, perhaps due to the fact that there are so many intermediate types of cases. Some neoplasms, from their histologic appearance, are clearly extremely malignant, and about these no difference of opinion exists.

The diagnosis of chorioepithelioma from the curettings alone is often impossible. Some specimens are so clearly malignant that a positive histologic diagnosis is entirely justifiable, but, unfortunately, many cases do not fall within this category. This point is appreciated by all gynecologic pathologists, but is not, I believe fully recognized by clinicians at large. It is my belief that not a few uteri have been removed because of a mistaken histologic diagnosis of chorio-

epithelioma, when the condition in reality was merely one of retained secundines. Moreover I believe that these cases often go to swell the numbers of the less malignant type of chorioepithelioma. I have encountered only six cases of chorioepithelioma. Three of these were of the manifestly malignant type and were readily diagnosed from the curettings.

In the entire series of 391 cases of malignant tumors the clinical diagnosis was positive and correct in 272 cases (69.3 per cent); the true condition was suspected in an additional 59 cases (15 per cent); in 15 (3.8 per cent) the clinical diagnosis was of malignant tumor, but the type of neoplasm was not recognized, and in 45 (11.5 per cent) the condition was clinically regarded as benign and its true character determined only on histologic examination.

The laboratory should work in cooperation with the clinic and should be closely associated with the latter, for their mutual benefit. The laboratory should have the benefit of clinical evidence in all cases, and the follow-up of the clinic should serve as a check on the laboratory diagnosis. In conclusion let me say that the most experienced pathologists may hold different views about certain specimens. Even after a most thorough study a certain percentage of cases will be encountered in which it is impossible to give a positive opinion. It naturally follows that the more experienced the pathologist, the smaller this group will be. As regards the question of whether or not any given case is malignant, it is my opinion that if, after a thorough study and examination of a number of sections, some doubt as to the malignancy still exists, these cases usually prove to be benign. However, the safe plan for the pathologist to adopt is to state plainly that he is in doubt, and suggest that the patient be kept under observation, and that a second biopsy or curettage be performed immediately upon the recurrence of any suspicious symptom. In other words, histologic diagnosis has definite limits, and this fact should be appreciated by the clinician. It is extremely unsatisfactory, from the clinician's standpoint, to receive such a diagnosis, but one positive diagnosis that later proves to be incorrect is far more unfortunate. A doubtful histologic diagnosis compels the surgeon to exercise his clinical judgment, which is generally excellent, and is far more preferable than to have him accept a doubtful diagnosis as positive and act accordingly.

CHESTNUT AND TWENTY-SECOND STREETS.

THE TREATMENT OF CYSTOCELE, RECTOCELE AND UTERINE PROLAPSE*

By Robert T. Frank, M.D., F.A.C.S., Denver, Colo.

BECAUSE after vaginal plastic repair, healing by primary intention is the rule and serious postoperative complications are infrequent, this intervention, like adenoidectomy, tonsillectomy, and curettage, is often performed by the general practitioner.

The indication for intervention is often based upon very doubtful foundation, much unnecessary operating being done. This is partly due to the fact that this region is given but scant notice in the standard text books of anatomy, and that proper anatomical material rarely finds its way into the dissection rooms, and even more so, to the fact that textbooks on gynecology concentrate their attention upon methods of denudation and suture rather than upon the proper selection of cases and the broad general principles involved in successfully narrowing the genital hiatus and retaining uterus, bladder and rectum in their normal relationship.

If properly approached, the essentials of the anatomy of the pelvic outlet can be demonstrated clearly and simply, and be learned and remembered without undue effort. This article aims to present the anatomy of the pelvis, the indications for intervention and the technic as based upon the anatomy, with the hope that it will prove of use to the profession and reduce both the number of operations performed unnecessarily and the large number of operative failures.[1]

ANATOMY

The pelvic outlet is closed in by a thin but strong musculofascial diaphragm, called the levator plate, composed of the two levatores ani and the two coccygeus muscles, which extend from the pubes to coccyx and sacrum (Fig. 1) and fuse in a median raphe.

Anteriorly, viewed from within, close to the inferior border of the symphysis, in the dissected subject, an elliptical gap is noted (known as the genital hiatus) (Fig 1). Through this opening the urethra and vagina pass downward. Behind the hiatus, and surrounded by fibers of the levator muscle, the rectum pierces the diaphragm.

The potential weak spot (the hiatus) is strengthened on its superficial aspect by a strong double membrane which closes up the subpubic angle by stretching from one descending ramus of the pubis to the opposite one (Fig. 2). This double fascia, containing between its

*Read at a meeting of the New York Obstetrical Society, October 10, 1922.

Fig. 1.—Pelvis, viewed from above. The levatores ani muscles and coccygei form the diaphragm. Behind the symphysis an elongated gap (genital hiatus) appears, through which the urethra and vagina pass. The puborectal fibers are shown projecting beyond the edges of the gap which appears unduly long as the rectum has been removed. The gap is reinforced below by the triangular ligament (see Fig. 2).

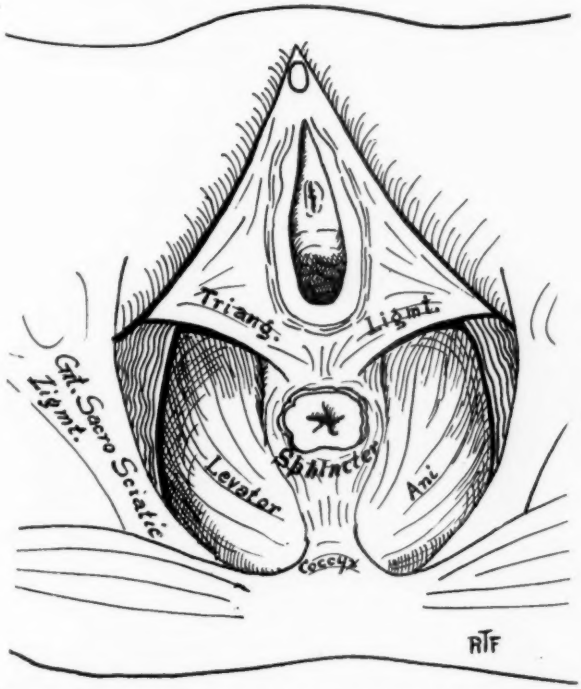

Fig. 2.—Pelvis of nullipara viewed from below. The skin, fat and superficial muscles have been cut away. The relations of the triangular ligament to the sphincter ani are well shown. The levator ani has been denuded of its outer (rectal) fascia.

superficial and deep layer the deep transverse perineus muscle (Fig. 3) is triangular in shape, and is therefore called the triangular ligament.

The genital hiatus is bordered on each side by fibers of the levator muscle, fibers which run from the posterior surface of the pubic bone to the rectum, and hence are known as puborectal fibers (Fig. 3). Along the free edge formed by the puborectal fibers two layers of fascia meet and fuse. The inner abdominal surface of the pelvic diaphragm (i.e., levator and coccygeus muscles) is clothed by a fascia called the levator fascia. The outer surface facing toward the ischiorectal fossa, is invested by a fascial layer named the rectal fascia (Fig. 9). Along the free margin of the genital hiatus, which corresponds to the edge of the puborectal fibers, the inner (levator) and outer (rectal) fascia meet and form a rounded edge (Fig. 6). To this edge the deep layer of the triangular ligament is applied, thus strengthening and closing a potential hernial site (Fig. 3).

The urethra and vagina penetrate both layers of the triangular ligament and during their passage through this structure receive fascial investments from it. The vagina also obtains some connection with the fascial edges of the genital hiatus which border its lateral walls.

Between the pelvic diaphragm below and the lowest portion of the peritoneal cavity above there is a considerable space.. This subperitoneal space is shallower anterior and posterior to the uterus, where the uterovesical and uterorectal (or Douglas') culdesacs encroach upon it, but deep laterally where it forms the bases of the broad ligaments (Fig. 4).

The subperitoneal space harbors the blood vessels, nerves and lymphatics which supply the pelvic organs, and also the pelvic portion of the ureter.

The space is filled mainly by a network of strong bands of white and yellow elastic connective tissue of immense strength, which contains fat and unstriped muscle within its meshes. The strands appear to radiate from the supravaginal part of the cervix and are inserted into the fasciae lining the pelvic walls (obturator, levator). The vagina and rectum are surrounded by this connective tissue, which forms, respectively, the paracolpium and paraprocteum. The base of the bladder rests upon the thin layer running from the cervix to the posterior border of the pubic bones, pubocervical ligaments, or fascia (Fig. 5). Lateral to the uterus the connective tissues form the strong and massive parametria.

This subperitoneal connective tissue plays the major rôle in retaining the pelvic viscera in position. Its elastic structure allows a limited degree of motion. Excessive, and especially sudden strains, force the viscera downward upon the pelvic diaphragm which, though thin,

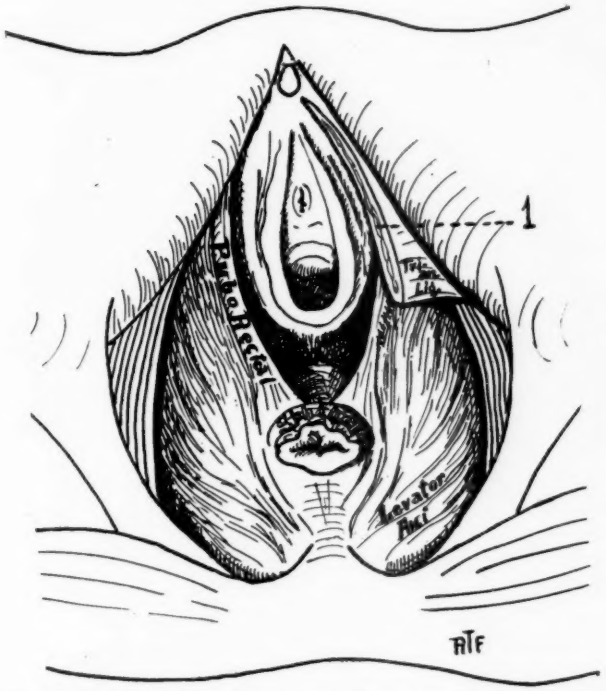

Fig. 3.—Pelvis of nullipara, viewed from below. The triangular ligament has been cut away except a small portion on the right. Vagina and rectum have been separated for a short distance. The puborectal fibers of the levator ani form the edge of the genital hiatus (or levator gap). Note the sphincter ani, superficial to the levator muscle.
t, Deep transverse perineus muscle appearing in cut section between layers of the triangular ligament.

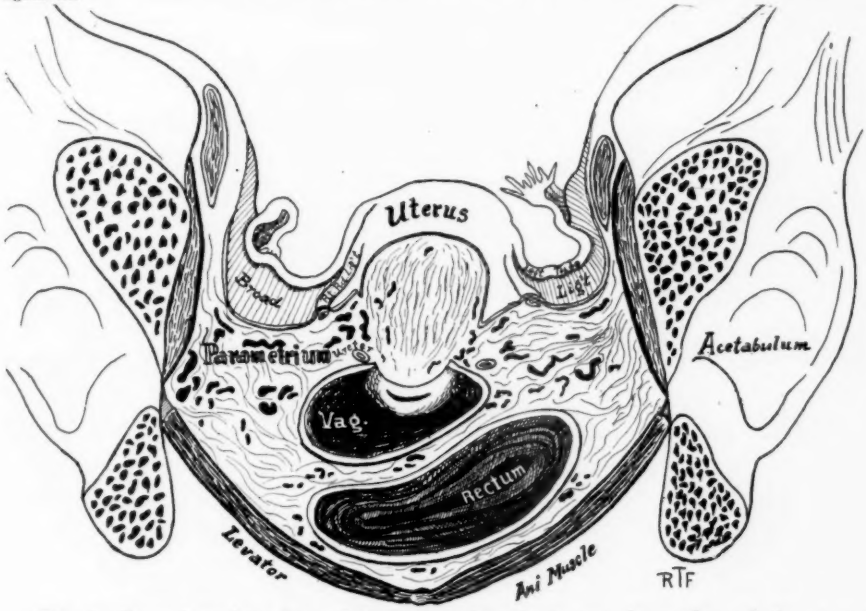

Fig. 4.—Coronal section of a pelvis, passing through the anterior surface of the uterus. Note the massive connective tissue lying between the peritoneum and the levator muscle. The latter stretches across the pelvis. Above the parametrium proper, is the thin, transparent broad ligament.

is powerful. Contraction of the levator group then not only produces an upward lift and shallowing of the bowl-shaped pelvic cavity, but also a narrowing of the genital hiatus, all tending to give immediate, powerful but transitory resistance to downward pressure.

The uterine ligaments, of which the round and sacrouterine ligaments are alone of importance in this connection, play an important rôle only so far as they serve to tilt the uterus and thus subject either the anterior or posterior surface of this organ to the forces of intraabdominal pressure.

Short round and sacrouterine ligaments favor anteflexion; relaxation encourages retroversion and retroflexion. The ligaments play

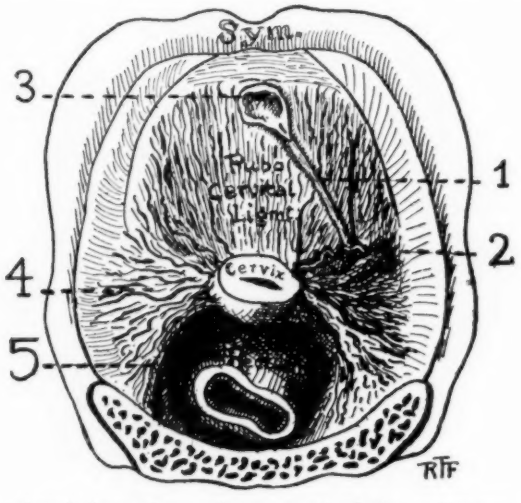

Fig. 5.—Pelvis viewed from above after removal of the peritoneum. The body of the uterus, the bladder and left ureter have been removed. The wedge-shaped parametria, sacrouterine folds, and thin anterior bladder shelf (pubocervical ligament) are shown.
1, Right ureter; *2*, uterine artery; *3*, urethra; *4*, parametrium; *5*, sacrouterine fold.

a most subsidiary rôle as far as fixation is concerned largely because they lack tensile strength.

PATHOLOGY

Childbirth causes injuries to the muscular, fascial and connective tissue structures of the pelvis. The triangular ligament may be torn, one or both of the puborectal loops of muscles may be avulsed from the pubic bones, the genital hiatus may be greatly dilated, the pubocervical fascia may be split, etc., etc. It is not always possible to define or recognize the tissues most injured. In general, however, three main types of injury result and can be recognized and corrected.

The evidence of these injuries appears either separately or combined as cystocele, rectocele and prolapse of the uterus.

Cystocele is a descent of the bladder, projecting downward through

the anterior vaginal wall, and due to sagging, separation, or rupture of the pubocervical connective tissue. Rectocele may be high, appearing as a bulging of the posterior vaginal wall, and descending sometimes over an intact perineal body (Fig. 9B), or low, due to a tear of the fascia investing the rectum, plus a torn perineum. The high rectocele is due to a tear of the fascia of the rectum high up, at the bottom of Douglas' culdesac and resembles a sliding hernia (Fig. 9).

Prolapse or descent of the uterus shows itself in various degrees. The cervix may descend below the level of the ischial spines (first degree), reach the level of the vulva (second degree), or the entire uterus may protrude beyond the vulva (third degree or total prolapse).

The main lesions invariably consist in a tearing, or more often, stretching of the pelvic connective tissues, especially the lateral parametria. Such factors as retroversion or flexion, elongation of the cervix, or tears of the perineum, if present, are merely concomitant but not causative. They are, however, often due to the same etiology, namely, childbirth.

Cystocele, rectocele and prolapse of the uterus may be present coincidentally. Elongations of the cervix, cervical lacerations, retroflexion and other complicating troubles may, too, require correction.

INDICATIONS FOR TREATMENT

Not every case of cystocele, rectocele and prolapse requires operative intervention. Many patients should at least be tided over their period of child-bearing activity by palliative measures.[1]

A cystocele alone, if the perineum is intact, may be retained by a Skene or a Gehrung pessary. This is necessary if frequency of urination or incontinence develops.

A rectocele, alone, rarely causes more than annoyance. No pessary in the long run will adequately retain it.

Prolapse of the uterus, in a young woman, who desires to have more children, may be kept back with simple pessaries such as the saucer pessary of Schatz, or the hard rubber ring, globe or egg-shaped pessaries. These pessaries are removed at night by the patient and reinserted before rising. If cleanliness and care are exercised, they may be used continuously for many years.

It is also unwise to operate upon old, debilitated women who are poor operative risks. Here, too, the pessaries just mentioned are advised.

If operation is to be performed upon a woman who expects to have more children, the vaginal tube and outlet must not be unduly narrowed. Care must be exercised in repair of the cervix, trachelorrhaphy according to Emmet's method being preferable to cervical

amputation or tracheloplasty (by coring), because amputation frequently induces sterility, or if pregnancy supervenes, may cause severe dystocia. If the uterine body is displaced, most frequently either retroflexed or retroposed, the corrective operations should aim to shorten the round ligaments and not fasten the uterus to the abdominal wall, because ventrosuspension or ventrofixation may produce serious disturbances throughout pregnancy and during labor.

In women, who are to be sterilized at operation, or who have passed

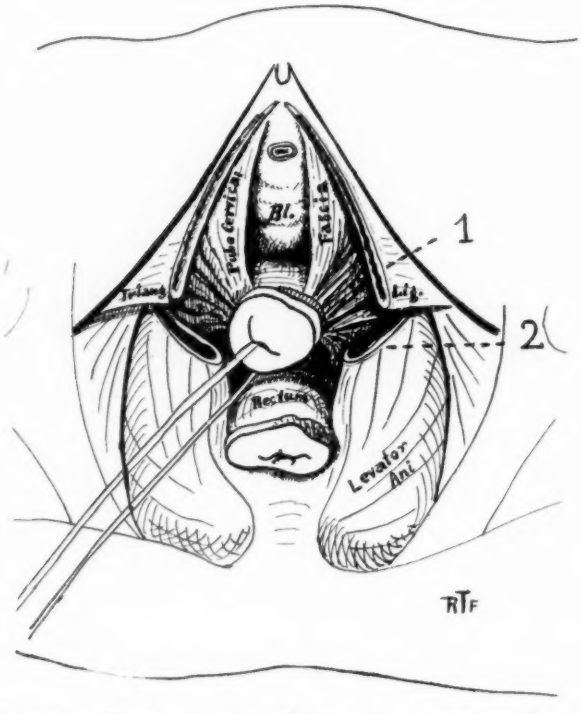

Fig. 6.—Pelvis of nullipara, view from below. The central portion of the triangular ligament has been removed and the upper portion of each levator muscle excised, in order to expose the cellular, connective tissues. By means of a suture through the cervix, the uterus is pulled down and to the left. The pubocervical fascia (or ligament) has been split sagitally to expose the bladder.

1, Appears to end on triangular ligament but points to the parametrium; *2*, edge of levator ani muscle cut across (in red) surrounded by its fasciae.

the menopause, none of the above objections apply. Here three methods of operation may be considered.

1. Repair of the anterior and posterior vaginal wall and perineum from below, followed by ventrofixation.

2. Interposition of the uterus between the inferior bladder wall and the vagina, and repair of the perineum.

3. Vaginal hysterectomy, with suture of the lateral stumps below the bladder, followed by perineal repair.

In very old women, or where severe recurrence of prolapse has taken place after vaginal hysterectomy, obliteration of the vagina may be practiced, if the patient and husband are first enlightened as to the end results of the operation.

With very few exceptions I have found the first method, repair of the cystocele and rectocele from below, combined with either the Alexander-Adams operation or ventrofixation from above, applicable to all varieties of prolapse.

Occasionally, in short fat women, where laparotomy is extra hazardous, and where a large cystocele causes the most serious disability,

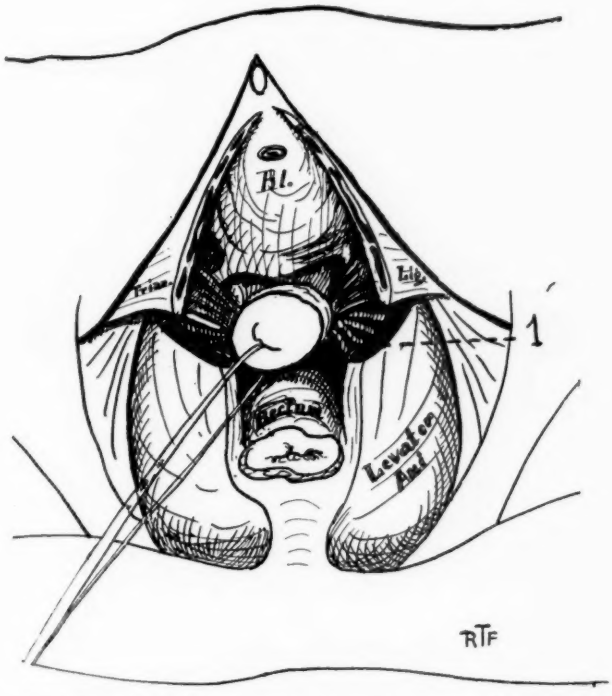

Fig. 7.—Further dissection of Fig. 6. The pubocervical fascia has been removed, exposing the bladder and showing the beginning of both ureters and their entrance into the parametria. *1*, Edge of levator ani muscle cut across.

I have used the interposition operation.[2] Even more rarely, in old women long past the menopause, who nevertheless possess a uterus of fair size, I have interposed the uterus, taking special care to fasten the uterine body to the pubocervical fascia and to the edges of the genital hiatus (Fig. 6).

I have never used the method of vaginal hysterectomy with utilization of the broad ligament stumps as described by Goffe,[3] and would reserve it for those rare cases of prolapse in which a vaginal hysterectomy is indicated for other reasons, as for example, in corporeal cancer. I have seen a number of cases in which this technic

was used, followed by huge recurrences of the cystocele. Such recurrent cystoceles may prove incurable, as the main central support of the pelvic connective tissues, from which the supporting fibers radiate, namely, the supravaginal part of the cervix, has been removed. This removal destroys most especially the pubocervical fascia or bladder shelf (Fig. 7).

Obliteration of the vagina (LeFort's operation) is indicated mainly

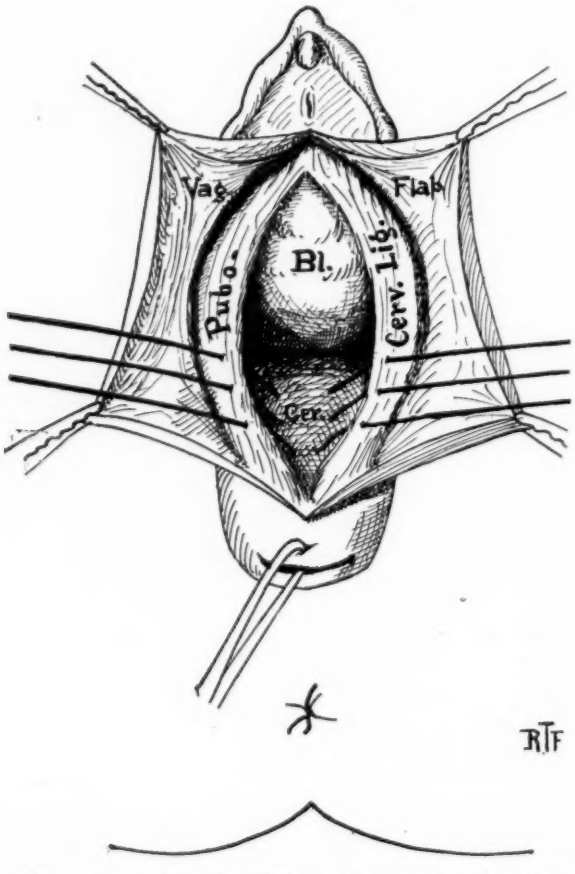

Fig. 8.—Operative exposure for the correction of cystocele. Vaginal flaps are retracted laterally. The pubocervical fascia (or ligament) is exposed and three sutures have been placed, which, when tied, shorten and raise the insertion of the fascia, thus elevating and retaining the bladder in a high position.

in the above cases and in very old and debilitated women, in whom a short intervention under local anesthesia is alone feasible.

The modern technic for repair of cystocele and rectocele by the methods to be described is the result of gradual evolution. The anatomical basis was given mainly by Hadra, Tandler, Halban and Martin, and others.[4] Unless the anatomy is fully understood, most uneven and unsatisfactory results will be obtained.

REPAIR OF CYSTOCELE

The principle involved is based upon separation of the descended bladder from the uterine cervix, repair of the pubocervical fascia in the median line, and suture of this structure high up to the cervico-uterine junction so as to reestablish a tense continuous bladder shelf.

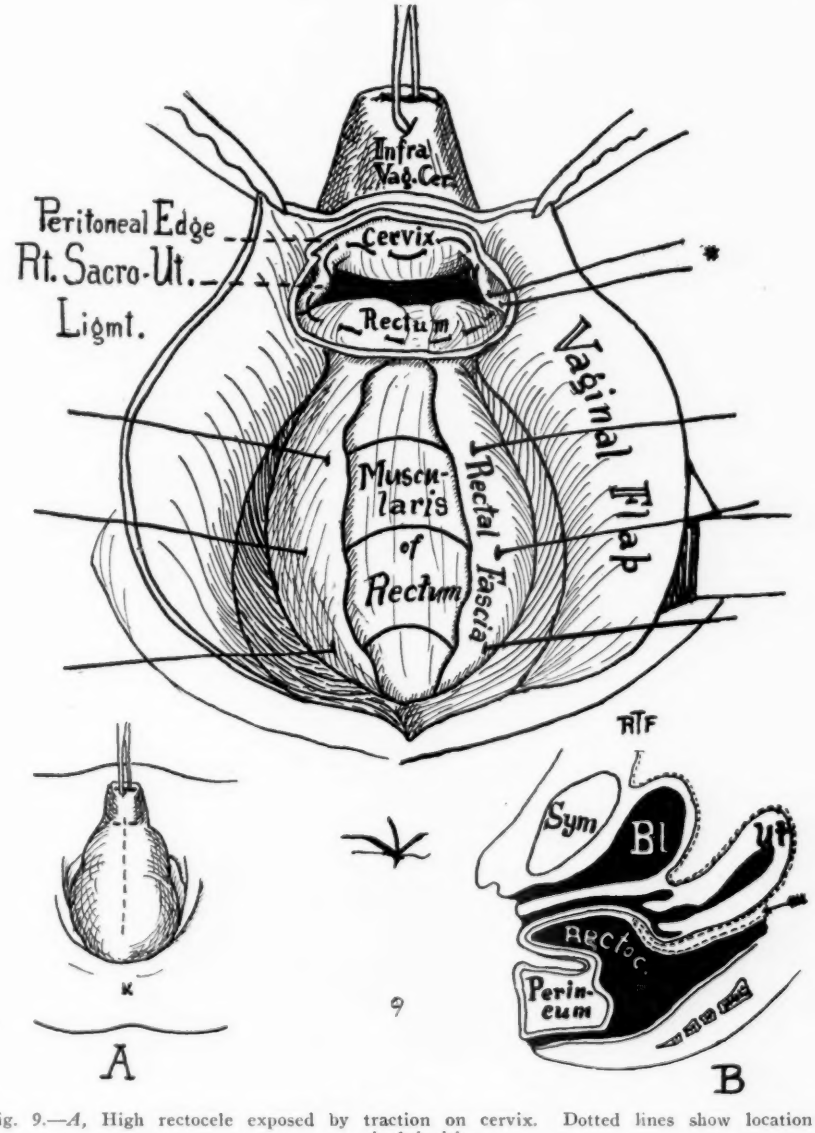

Fig. 9.—*A*, High rectocele exposed by traction on cervix. Dotted lines show location of vaginal incision.

B, Schematic median sagittal section showing relation of high cystocele to the culdesac, posterior vaginal wall and intact perineum.

C, Operative exposure of high rectocele. Vaginal flaps have been retracted, the torn rectal fascia exposed, and above, Douglas' culdesac has been opened. An asterisk marks the ends of the linen suture which obliterates the culdesac. Below are three sutures ready to be tied to close the rent in the rectal fascia.

The cervix is grasped with a volsellum and pulled down. A second forceps is placed ¼ inch below the external urinary meatus. The anterior vaginal wall is lightly incised between these two instruments. Two vaginal flaps are separated, care being taken to stay immediately beneath the vaginal mucosa. The tissue between bladder and cervix is now snipped in the median line and the bladder bluntly pushed off from the cervix *in the median line only*. If these directions are observed, strong fascial bands will be demonstrated running from the cervix upward and disappearing behind the posterior surface of the symphysis. These fibers are the pubocervical fascia (Fig. 6), continuous laterally with the parametria (Figs. 6 and 7). After defining this fascia, the bladder may be freely pushed off the cervix mesially and laterally. Before inserting chromic gut sutures through the fascia, cervix, and fascia on the other side, as shown in Fig. 8, careful hemostasis must be performed, as a retrofascial hematoma is usually fatal to success. The interrupted sutures are tied, thus retaining the bladder above the fascial shelf. Any part of the fascia not approximated is closed by additional sutures. The edges of

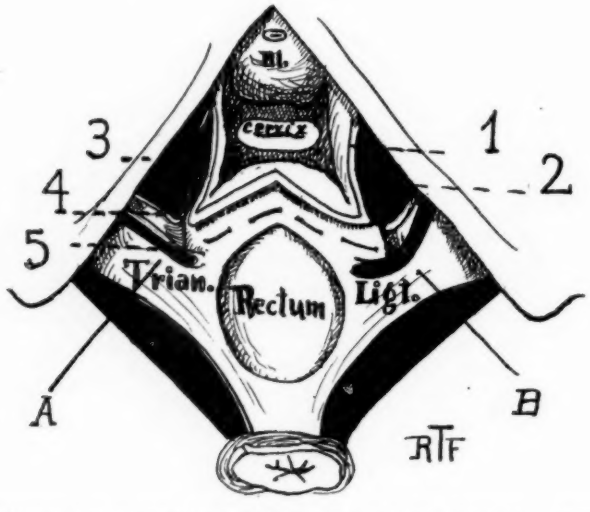

Fig. 10.—Dissection of a multiparous pelvis showing a low rectocele. Part of the vagina has been cut away, as well as the upper part of the stretched and torn triangular ligament and part of the rectal fascia. Note the structures traversed by the purse string suture *A B*. *1*, Vaginal edge; *2*, levator edge; *3*, levator ani muscle; *4*, rectal fascia; *5*, edge of deep transverse perineus muscle cut across.

the vaginal flaps may be trimmed, to avoid redundancy, and the wound in the anterior vaginal wall closed by interrupted or continuous suture of chromic catgut or silk.

If curettage, trachelorrhaphy or amputation of the cervix are to be performed, these operations should precede the anterior repair.

REPAIR OF HIGH RECTOCELE

Exposure of the anterior rectal wall high up, opening of Douglas' culdesac, obliteration of this pouch by circular suture and repair of the torn rectal fascia. This repair is usually combined with repair of low rectocele and torn perineal body.

The posterior lip of the cervix is grasped and the uterus pulled upward and forward (Fig. 9-*A*). Immediately below the bulging portion of the rectum (cor-

responding in most instances to about 1½ to 2 inches within the vulva) a volsellum grasps the lateral posterior sulcus on each side. A median incision is made over the bulging rectum reaching to the top of the posterior fornix (Fig. 9-*A*). Two vaginal flaps are reflected, mainly by blunt dissection, and keeping close to the vaginal mucosa, until the edge of the torn and retracted rectal fascia is encountered on each side.

If now it appears as if the rectum were projecting downward from behind the cervix, in the fashion of a sliding hernia, the posterior culdesac is opened by a transverse incision through the peritoneum behind the cervix. Each peritoneal edge (anterior and posterior) is temporarily secured by a guide suture.

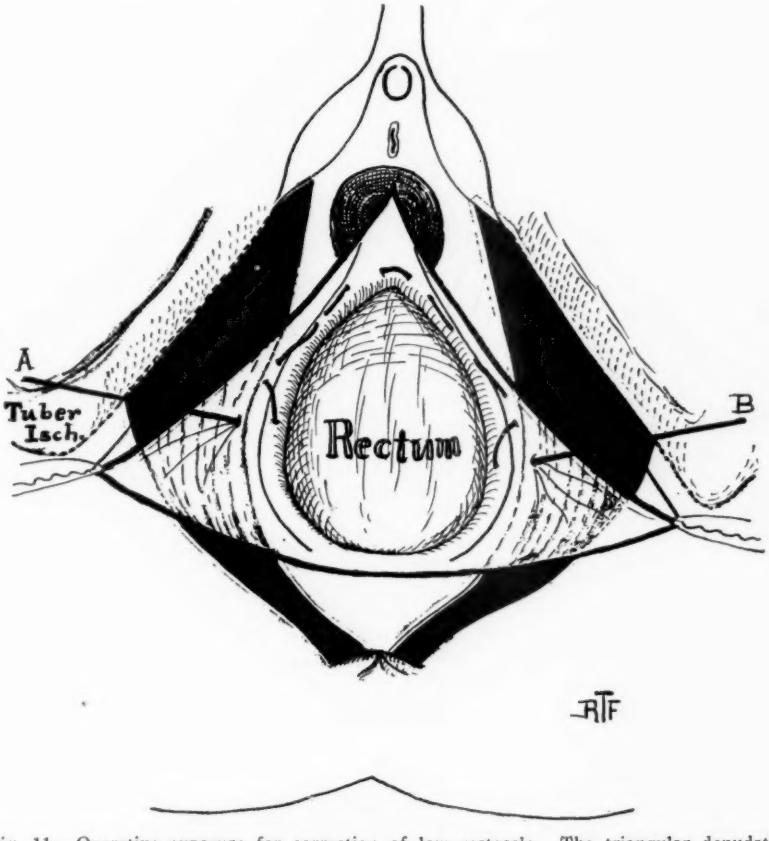

Fig. 11.—Operative exposure for correction of low rectocele. The triangular denudation is shown in line, the levator ani within this area is indicated uncolored, as if the tissues were transparent. Compare with Fig. 10 and note that the purse string suture *A B* traverses the triangular ligament, levator muscle with its fasciae (rectal and levator fasciae) and the fascia propria of the rectum on each side.

A purse string linen suture is now passed intraperitoneally, beginning on the patient's left side and taking in the left sacrouterine ligament as high up as can be reached, then passed through the peritoneal and muscular coats of the anterior rectal wall in at least three places, next taking the right sacrouterine ligament, and passing firmly in the cervicouterine junction (posterior cervical wall) and ending again in the left sacrouterine ligament (Fig. 9). This purse string is tied, thus obliterating the base of Douglas' culdesac, and, if properly placed, pulling the rectum upward. The peritoneal opening may be separately closed.

The rectal fascia which has been clearly defined by blunt dissection is approximated by a continuous suture of fine chromic catgut. The vaginal wall is trimmed and sutured with interrupted stitches to within an inch of the lower angle of the wound.

REPAIR OF LOW RECTOCELE AND LACERATED PERINEUM

Exposure of the anterior rectal wall, separation of the rectum laterally from adjacent structures, approximation of the levator edges and triangular ligament en masse.

The beginner will do well to seize the crest of the rectocele at a point which can

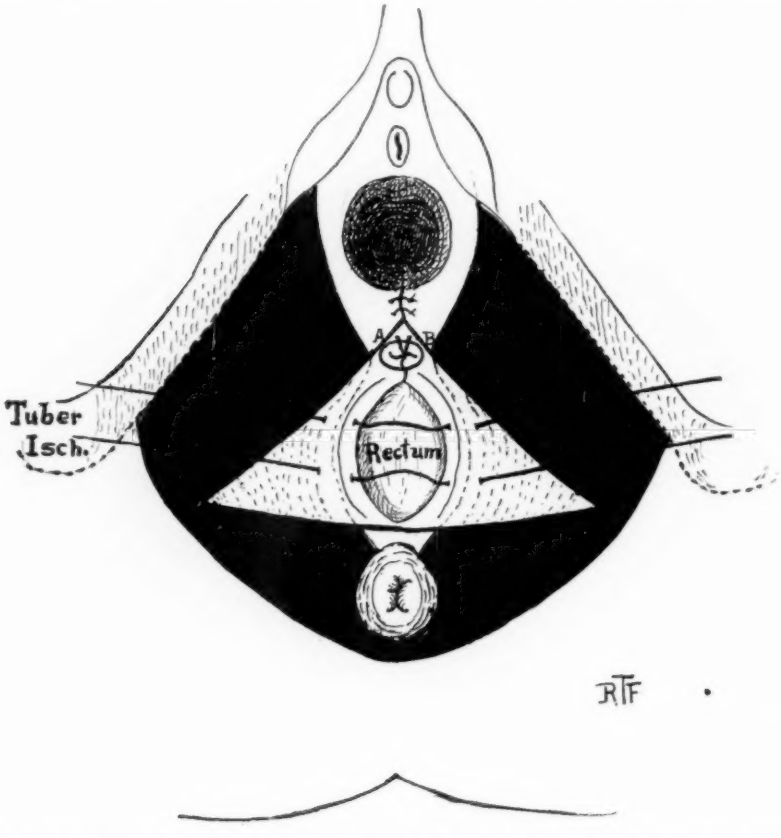

Fig. 12.—Fig. 11 after the purse string suture *A B* has been tied. The genital hiatus has been narrowed, the triangular ligament tensed, the anus elevated. Two sutures which traverse from side to side have been placed. When tied they will close the gap remaining.

be readily raised to the level of the external urethral meatus (Fig. 11). Laterally, points above the outlet of Bartholin's duct are grasped and a small triangle of vaginal mucosa denuded. A median incision gives less convenient access, but, if preferred, can be used.

The anterior rectal wall is thus exposed. Laterally toward the perineum, the gloved finger covered with gauze can bluntly push the rectum medianwards away from the tissues, taking pains not to penetrate into the deeper fascial layers. This blunt dissection, which occasionally tears large, friable hemorrhoidal veins requir-

ing no separate ligation, exposes on each side a triangular area containing the edge of the triangular ligament and, more deeply and laterally, the levator edge and its investing fasciae (Fig. 10).

Penetration of these deeper layers serves to expose the deep transverse perineus muscle (often mistaken for the levator ani) and the levator. The muscle fibers, deprived of their fascia, have little tensile strength, and the resulting perineum is scarry and tender. Hence I have long since abandoned suture of the exposed muscle fibers.

By gauging the mobility of the lateral structures, and visualizing the degree of approximation sought for the levator edges (see Fig. 12), the site for the purse-string suture to be placed can be determined. The beginner can seize the tissues tentatively with volsellum forceps and test the result by crossing these instruments.

A heavy chromic catgut suture is passed deeply on the patient's left (Fig. 11) grasping levator edge and triangular ligament, coming out close to the rectal wall. Light bites of the torn rectal fascia are taken until the crest is reached, the rectal fascia of the opposite side being gathered in turn. When a point opposite to the deep part of the suture has been attained, the tissues on the patient's right are penetrated in a reverse direction, see Fig. 11. A.

The tying of this purse string not only brings together the levator edges and triangular ligament but also raises the anus back to its proper position from which it has sagged (Fig. 12). The small remaining gap in the rectal fascia can be closed with interrupted chromic catgut sutures. The subcutaneous tissues and vaginal edges are then approximated. A subcuticular suture for the skin edge, reinforced by skin clips favors primary union.

If repair of a high rectocele preceded the low repair, the apex of the lower denudation begins approximately at the lower margin of the upper wound area.

SUMMARY

If the anatomy of the pelvic outlet is understood, if cases are judiciously selected, and if the technic described is followed, the results are fully as satisfactory as those obtained in the radical cure of inguinal hernia. A certain number of recurrences may be expected, especially in patients with flaccid tissues and general enteroptosis. It should be emphasized that patients, whose complaints and pains did not arise from the minor lacerations of the cervix, the small cystocele, the negligible rectocele present, will not be benefited by unnecessary plastic repair.

CONCLUSIONS

1. The bony pelvis is closed in by a bowl-shaped musculofascial diaphragm, the levator plate.

2. This diaphragm in its anterior portion has an elliptical gap, the genital hiatus, bordered laterally by the levator edges.

3. The genital hiatus is partially closed and the diaphragm is reinforced by the triangular ligament which extends from one pubic ramus to the other. The urethra and vagina pierce the triangular ligament.

4. The uterus, bladder and rectum are retained in their normal positions by strong, yet elastic, bands of connective tissue and un-

striped muscle, most important of which are the parametria, pubocervical fascia, paraprocteum and paracolpium.

5. The pelvic diaphragm takes up exceptional and especially short, excessive strains and stresses. Alone it is unable to support the pelvic viscera.

6. The round ligaments and sacrouterine ligaments possess little tensile strength and serve merely to tilt the uterus forward or backward.

7. Injuries, resulting almost exclusively from childbirth, produce defects in the pelvic tissues which cause cystocele, rectocele and prolapse.

8. Careful selection of cases requiring intervention must be made. Many patients with minor injuries require no operation. Patients still bearing children, may be, and the old and debilitated should be, treated palliatively.

9. During the age of childbearing, plastic vaginal repair must not narrow this canal to excess, and the uterus must be antiflexed by shortening of the round ligaments.

10. After the menopause, or after sterilization, ventrofixation may be used to aid in sustaining the uterus.

11. The repair of cystocele and of high and low rectocele closely resembles the operations in vogue for cure of abdominal hernias. The main reliance is placed upon the closure of tears in the fascial structures and narrowing of preexistent gaps (the genital hiatus).

12. Other technics (vaginal interposition, vaginal hysterectomy, obliteration of the vagina) should be reserved for very special and exceptional circumstances.

BIBLIOGRAPHY

(1) *Frank, R. T.:* A Study of the Anatomy, Pathology and Treatment of Uterine Prolapse, Rectocele and Cystocele, Surg., Gynec. and Obst., 1917, xxv, 42.

(2) In America, *Watkins* has fathered the interposition operation. (Am. Gynec. and Obst. Jour., 1899, xv, 420; Surg., Gynec. and Obst., 1917, xxv, 147.) In Europe, *Wertheim, E.* (Zentralbl. f. Gynäk., 1899, xx, 1009) and *Schauta* (Beitr. z. Geburtsh. u. Gynäk., 1903, xi, 68) were its main advocates. A fairly high mortality rate of 3.4 to 7.5 per cent and recurrences in 7 to 20 per cent speak against the routine use of this method (for statistics see *Döderlein and Krönig*), Operative Gynäkologie, ed. 4, Thieme, Leipzig, 1921, p. 320-321).

(3) *Goffe, J. R.:* An Improved and Perfected Operation for the Relief of Extreme Cases of Procidentia, Cystocele and Rectocele, Am. Jour. Obst., 1910, lxii, 611.

(4) *Hadra, B. E.:* Remarks on Vaginal Prolapse, Rectocele and Cystocele. Am. Jour. Obst., 1889, xxii, 457. (This author recognized the importance of the pelvic connective tissues in prolapse and also advocated freeing the bladder for cystocele.)

Freund, H. W.: Über moderne Prolapsoperationen, Centralbl. f. Gynäk., 1901, xxv, No. 18, p. 441, also advocated liberation of the bladder and its retention by means of the paravaginal tissues.

Halban, J., and Tandler, J.: Anatomie und Aetiologie der Genitalprolapse beim Weibe. Braumüller, Wien 1907, ascribed the sole supportive properties to the musculature, especially the levator ani.

Martin, E.: Der Haftapparat der weiblichen Genitalien, Karger, Berlin, 1911 and 1912, placed prime importance on the connective tissues.

523 MAJESTIC BLDG.

(*For discussion see p.* 93.)

OBSERVATIONS ON THE BLOOD SUGAR DURING PREGNANCY AND THE PUERPERIUM

By Walter N. Rowley, M.D., Rochester, Minn.

From the Section on Obstetrics, Mayo Clinic

THE sugar concentrations of the venous and cord bloods were studied in a series of twenty pregnant women in order to compare the blood sugar concentration in the maternal blood at or near term with that thirty-six to forty-eight hours after delivery, at a time when the factors of shock or anesthesia can be excluded but when the uterus is still in the process of involution. The cord blood in every case was taken at the time of delivery.

The method of obtaining the specimen of blood from the mother was identical with that used routinely in the clinical laboratory for collecting blood chemistry specimens. The blood was taken about 8 A.M., the last meal having been served at 6 P.M. the day before and no breakfast allowed until the blood had been drawn. This allowed a fasting period sufficiently long to eliminate the influence of alimentation on the blood sugar concentration.

The median basilic vein was punctured and a turniquet applied to the arm. The blood was collected in sterile tubes containing enough potassium oxalate to prevent clotting. This specimen was analyzed by the modified Benedict method and by the same technicians who have charge of all the clinical blood chemistry. The patients were bled during the later months of pregnancy, usually at term or when they entered the hospital preceding labor, and again on the second morning after delivery, about thirty-six to forty-eight hours postpartum. The cord blood was obtained at delivery. As soon as the child had been delivered and properly cared for, the umbilical cord was clamped close to the abdomen and vulva, and the blood thus isolated from the cord was collected in a tube.

None of the patients in this series showed signs or symptoms of diabetes during antepartum care. Glycosuria was not present at any of the bi-weekly examinations of the urine.

The importance of blood sugar studies in pregnancy has been recognized. Schirokauer, Bergsma, Kampf, Benthin, Neubauer and Novak, Morriss, Slemons, Schiller, Killian and Sherwin, Guy, Caldwell, and others have contributed to the literature. Much of the work of foreigners was done by methods other than that of Benedict. Table I shows the comparative values noted by various observers.

Bergsma made numerous observations by the Frank-Moeckel plasma

method of sugar determination, and he asserts that it probably gives slightly lower values than the Benedict method. He found a maximal value of 0.136 per cent for the sugar concentration during the later months of pregnancy and a mean value of 0.09 per cent. The time of drawing the blood was not the same in all his cases. In the group of cases here reported, however, the patients were bled with uniform consistency both in time and method. Blood taken during the puerperal period gave the same value as during normal pregnancy. Blood from the cord had an average sugar concentration of 0.11 per cent. This, however, was influenced by anesthesia in some cases.

Morriss gives a chart of the comparative blood sugar values obtained by seven other observers who studied the blood sugar values during pregnancy. His own figures show a mean value of 0.10 per cent. The general average range of the normal may be considered as from 0.09 to 0.11 per cent. These percentages agree with findings in normal nonpregnant women. According to Morriss, the blood sugar content of cord blood was 0.12 per cent at birth and 0.13 per cent at the end of the second stage of labor. In nineteen of twenty cases, the maternal value at the end of labor was higher than the fetal value for blood sugar. Morriss' percentages show that the fetal blood is higher than the maternal blood during pregnancy. The factors in these calculations, however, were not consistent or constant and make the values less significant. Variations in the fetal blood were observed in cases in which an increase was noted in the maternal blood as a result of a long second stage labor, prolonged anesthesia, or operative delivery. In such cases Morriss found the mean value for the cord blood readings to be 0.17 per cent as compared to 0.13 per cent in the normal.

Schiller found values within the normal range for blood sugar concentration during pregnancy. Killian and Sherwin's average was 0.11 per cent. Caldwell and Lyle found a mean value of 0.09 per cent at the end of normal labor, an average somewhat lower than that of Morriss. The introduction of anesthesia always augmented the values as did operative procedures and protracted labors. McGuigan and Ross, and Steward and Rogoff have shown that anesthesia is a definite factor in the production of hyperglycemia.

Scott and Hastings studied the sugar concentration in the blood of dogs during exercise and found that the values fell steadily throughout the work period. In patients whose blood sugar values were taken at the end of labor the concentration was higher. This suggests that Morriss' percentages for the second stage may have been principally influenced by the chloroform used in anesthesia. However, he demonstrates a greater concentration in primipara than in multipara during

labor, probably owing to the amount of voluntary effort put forth during the second stage.

The blood sugar values on the infant have been thoroughly investigated by Sedgwick and Zeigler, and Guy. The former obtained an average of 0.08 per cent blood sugar in infants ranging from three to forty-three days of age. Guy notes a range of 0.06 to 0.11 per cent in normal infants, with an average of 0.07 per cent in forty-three cases. The fetal blood shows variations as a result of anesthesia, long labor, operative procedure, and asphyxia. Morriss' data show the influence of these factors on the fetal blood values which he found to rise when the maternal blood value rose.

The data which have been accumulated in my observations do not fall wholly in accord with the findings recorded in the literature. A series of fifty-three patients were observed during pregnancy, and a mean blood sugar value of 0.11 per cent was noted. The maximal value was 0.25 per cent in one instance; unfortunately, the postpartum reading was missed in this patient. No signs of diabetes were found, and labor was spontaneous and normal. The cord blood values determined on twenty-two specimens gave an average of 0.09 per cent. Thus, my data show an average maternal value of 0.11 per cent and a fetal value of 0.09 per cent; this definitely points to the fact that the maternal to fetal ratio is such that placental interchange of glucose can take place by a process of diffusion from the maternal to the fetal side. The mean average for the blood sugar concentration of normal pregnancy in this series approximates the upper limit of the general average which is 0.09 to 0.11 per cent. The fetal value of 0.09 per cent is slightly lower than the general average others have obtained, although this is the only group of readings influenced in any way by anesthesia, asphyxia, voluntary muscular exertion, or operative procedures.

Thirty-two blood sugar determinations were made on the cord blood. The highest reading, 0.15 per cent, was obtained in the case of a child (A230406) delivered by low forceps, the indications being fetal asphyxia. This would account for the hyperglycemia in this child even though ether, which was the anesthetic in all cases, was not taken into consideration.

The series of blood sugar determinations made postpartum was obtained on the morning of the second day after anesthesia, and so was not influenced by extraneous factors. Twenty-two cases were observed and a mean average of 0.14 per cent obtained; this is also higher than that obtained by other observers.

In twenty patients it was possible to obtain consecutive observations of the blood sugar during pregnancy, two days postpartum, and on the child from the cord blood. These patients have been divided

TABLE I

COMPARATIVE BLOOD SUGAR VALUES

	NORMAL PREGNANCY	FIRST STAGE LABOR	SECOND STAGE LABOR	PUERPERIUM	CORD BLOOD, NORMAL LABOR	EARLY INFANCY	OPERATIVE MATERNAL	LABOR FETAL	ANESTHETIC
Bergsma	0.09 (plasma)			0.09	0.11				Chloroform
Morriss	0.103	0.093	0.13	0.11	0.12		0.17	0.13	Chloroform
Sedgwick and Zeigler						0.08			
Schiller	0.10								
Caldwell and Lyle				0.09		0.09			
Killian and Sherwin	0.11								
Guy						0.07			
Rowley	0.11			0.14	0.09				Ether

TABLE II

GROUP 1. BLOOD SUGAR CONCENTRATION AUGMENTED DURING PUERPERIUM

CASE	AGE	PARITY	GAIN IN MOTHER'S WEIGHT, PER CENT	CHILD'S WEIGHT AT BIRTH		HEMORRHAGE POSTPARTUM	BLOOD SUGAR VALUE			CHARACTER OF LABOR
				POUNDS	OUNCES		BEFORE LABOR	AFTER LABOR	CORD BLOOD	
A335823	30	4		7			0.10	0.13	0.10	Forceps
A351603	32	1	22	7	1	Slight	0.08	0.14	0.10	Forceps
A351287	20	1	25	6	14	Moderate	0.12	0.17	0.08	Spontaneous
A75224	29	1	24	7			0.10	0.13	0.06	Forceps
A351390	27	3		8	8	Moderate	0.09	0.10	0.08	Spontaneous
A355413	25	2	23	6	3.5	Slight	0.10	0.15	0.09	Spontaneous
A355294	27	2		8	13	Slight	0.11	0.14	0.09	Forceps
A295053	32	3	28	7	6.5	None	0.09	0.16	0.09	Forceps
A142719	35	2	18	6	2.5	None	0.13	0.16	0.08	Spontaneous
A359824	24	1		6	11	None	0.09	0.09	0.06	Spontaneous
A155870	40	3		8	9	Slight	0.07	0.14		Spontaneous
A346300	23	3	20	5	8	Slight	0.12	0.15		Spontaneous
Average			23				0.10	0.14	0.08	

into Groups 1, 2, and 3, according to the respective blood sugar concentrations. In Group 1, are the patients whose blood sugar concentration was augmented during the puerperium; in Group 2, are the patients whose blood sugar concentration was decreased during the puerperium, and in Group 3, are the patients whose blood sugar concentration remained constant. Table II contains data on the cases in Group 1, including age, parity, percentage gain in weight, blood lost postpartum, character of labor, blood sugar values, and child's weight at birth. The average value for the blood sugar during pregnancy in patients in Group 1 was 0.10 per cent, very close to the average for all the cases during pregnancy, 0.11 per cent.

The blood sugar concentration for the postpartum period in Group 1 is 0.14, a difference of 0.04. Table II shows clearly this difference in every case and the average difference in concentration is striking. Throughout the whole series this ratio exists, though some cases are reversed with small differences and others show no change. The alteration of the blood sugar concentration with a resultant hyperglycemia in the postpartum period noted by observers on the same individual make the findings more significant. The mean average of the cord blood sugars is 0.08 per cent, or 0.02 per cent less than the average concentration during pregnancy. This corroborates the findings of Morriss whose mean difference, however, was based on the value obtained at the end of labor. The influence of ether on the cord blood values in my series is negligible. In one case (Case A335823), the fetal value equalled the maternal value during pregnancy, and in another case (Case A351603), in which labor was long and forceps necessary, it was slightly higher. The mean values undoubtedly show that the placental exchange is dependent on the higher concentration of glucose in the maternal blood. The cord blood values in patients in Group 1 did not show the patient's response to the effects of anesthesia or muscular effort in the same proportions as in Morriss' series.

Age, parity, and change in body weight do not influence the blood sugar concentration; reduction in blood volume due to postpartum hemorrhage is of no significance. Glycosuria was not present in any of the patients or related in any way to the determinations. The fetal weight had no bearing on the determinations from the cord blood. Long labors did not influence the readings of the maternal blood; such an influence would no doubt be transitory and readjustment would have taken place by the morning of the second day. This also applies to anesthesia.

One patient in Group 1 (Case A351287) had an enlarged thyroid and a basal metabolic rate of +13 in the last month of pregnancy. This may have been a factor in the high blood sugar concentration

in the postpartum stage. [This is suggested by a patient (Case A346385) noted in Group 3.] In the first five patients in Group 1 delivered by low forceps, labor was exhausting. The postpartum blood sugar values in these cases, however, were not the maximum for the series. Three patients were delivered after thorough treatment for tertiary syphilis. This was not a factor in any way.

The cases in Group 1 show an average gain in weight of approximately 23 per cent during pregnancy; with one or two exceptions, the percentage increase was very close to this figure. There may be some significance in the loss of weight which occurs so rapidly on delivery of the child and secundines and continues by the elimination of fluids from the tissues, clinically noted by the large output of urine during the first day or two postpartum. This concentration of the body fluids may possibly increase the blood sugar values immediately postpartum.

In one patient studied since the compiling of Table II, repeated blood sugar determinations were made in the postpartum period. Glucosazone was found in the urine in the eighth month of pregnancy, and the blood sugar at that time was 0.08 per cent. On the second day postpartum, the reading was 0.10 per cent, on the fifth day 0.10 per cent, and on the seventh day 0.14 per cent. The concentration was consistently higher during the period of involution than during pregnancy.

Group 2 contains four cases in which the blood sugar concentration was decreased during the puerperium (Table III). The influences and factors of the specific case may account for the differences in concentration, though the general conditions are the same as in Group 1. In Case A230406 there is so little difference that the readings could almost be considered equal. In one case (Case A335129) the value for pregnancy, 0.17 per cent, is explained by nothing in the history or examination. The fetal blood value here is the result of a long labor and anesthesia, although the rise is slight when the length of anesthesia is considered. The high fetal value noted in Case A230406 is further proof of the influence of asphyxia on the blood concentration. The child was badly asphyxiated at birth and the blood sugar determination was made from blood taken from the cord immediately after delivery. The blood concentration in this group of patients is high throughout. Although the differences are the reverse of what they are in Group 1, they are very small.

Group 3 contains four cases in which the blood sugar values were the same during and after pregnancy (Table IV). The maternal sugar concentration was higher than the fetal value in every instance. Why the values were the same during and after pregnancy is not clear. In Case A346385 the thyroid was the controlling factor; the

TABLE III

GROUP 2. BLOOD SUGAR CONCENTRATION DECREASED DURING PUERPERIUM

CASE	AGE	PARITY	GAIN IN MOTHER'S WEIGHT, PER CENT	CHILD'S WEIGHT AT BIRTH		HEMORRHAGE POSTPARTUM	BLOOD SUGAR VALUE			CHARACTER OF LABOR
				POUNDS	OUNCES		BEFORE LABOR	AFTER LABOR	CORD BLOOD	
A348445	25	3		7	13	Moderate	0.13	0.11	0.06	Spontaneous
A335129	27	1	18	7		Moderate	0.17	0.13	0.11	Mid forceps
A230406							0.14	0.13	0.15	
A361293	25	1	22	6	12		0.11	0.09	0.05	

TABLE IV

GROUP 3. BLOOD SUGAR CONCENTRATION CONSTANT BEFORE AND AFTER LABOR

CASE	AGE	PARITY	GAIN IN MOTHER'S WEIGHT, PER CENT	CHILD'S WEIGHT AT BIRTH		HEMORRHAGE POSTPARTUM	BLOOD SUGAR VALUE			CHARACTER OF LABOR
				POUNDS	OUNCES		BEFORE LABOR	AFTER LABOR	CORD BLOOD	
A346385		6	10	5	12	Slight	0.17	0.17	0.05	Spontaneous
A351390	27	3		8	8	Moderate	0.09	0.09	0.08	Spontaneous
A305926	20	1	19	7	11	Slight	0.10	0.10	0.09	Spontaneous
A54202	26	1	35	7	8	Slight	0.14	0.14	0.11	Low forceps

gain in weight was only 10 per cent. The basal metabolic rate was +35 at the sixth month of gestation and +16 at the seventh month. The influence of hyperthyroidism on hyperglycemia is well established, the increased activity of the thyroid early in the postpartum period and its tendency to keep the blood sugar level high may account for the values obtained. In Case A351287 (Group 1) the influence of thyroid activity on the puerperal blood sugar value is apparent.

When the total figures are computed for all of the cases in the three groups and all other values obtained, the average maternal value during pregnancy is 0.11 per cent and the average fetal value 0.09 per cent. The blood sugar value obtained during pregnancy is less than that of the maternal blood postpartum and greater than that of the cord blood. This consistency in the data is most striking. Why this relationship exists in the majority of cases is not easily explained. The factors influencing blood sugar observations in the work of other investigators have been eliminated. Muscular exertion during labor is not a factor in the production of postpartum hyperglycemia. Anesthesia is a contributing, but not a great, factor in producing a rise in the cord blood values; asphyxia produces a more marked rise than anesthesia. Placental interchange undoubtedly depends on the difference in concentration in the maternal and fetal bloods.

TABLE V

BLOOD SUGAR CONCENTRATION IN PATIENTS WITH MARKED NAUSEA AND VOMITING

CASE	BLOOD SUGAR	CASE	BLOOD SUGAR
A335823	0.10	A324486	0.11
A354072	0.10	A130639	0.14
A353467	0.12	A335129	0.17
A352102	0.10	A335823	0.10
A47612	0.14	A347329	0.17
A365260	0.12	Average	0.12

Whether or not involution of the uterus has a direct bearing on this difference in blood sugar concentration is a matter of conjecture. Involution, however, involves many factors and not only consists in reduction of the size of the uterus and absorption and elimination of the products of destruction, but also involves great vascular and cellular changes in the whole body plus the establishment of a new function, that of lactation. The findings in one case not included in this series point against involution of the uterus as a factor influencing the blood sugar concentration. Determination of the blood sugar before labor showed a concentration of 0.11 per cent. Labor was not allowed to progress after onset and delivery was by cesarean section followed by subtotal hysterectomy. On the third day postpartum, the blood sugar concentration was 0.13 per cent and on the

sixth day 0.12 per cent. There is, however, a certain metabolic change early in the puerperium which influences the blood sugar concentration. Whether or not it is of the same origin as that which augments the glycemia of patients who vomit early in pregnancy has not been determined.

A series of eleven cases in which nausea or vomiting or both were prominent symptoms shows a consistently higher average for the blood sugar at this time than is noted during normal pregnancy (Table V). The blood sugar determinations were made on blood taken before the inauguration of any treatment other than that by fluids. The individual values are high in several instances and all are at or above the upper limit of the blood sugar fluctuation. The average for the eleven cases was 0.12 per cent. In one patient (Case A133697) who did not have nausea and vomiting but whose prominent symptom was anorexia of ten days' duration, the value obtained was 0.07 per cent. Dehydration was marked but this did not affect the concentration of sugar in the blood. The patient was very thin naturally and, having taken no food for ten days and but little water, she lost a great deal in weight. Concentration of body fluids, as a cause for the higher values obtained in patients with vomiting, seems to be ruled out by the case in which dehydration was the prominent feature. In this group, the toxic factor producing the condition seemed to influence the glycemia. Killian and Sherwin found an average blood sugar value of 0.13 per cent in patients with nephritic toxemia and 0.12 per cent in those with hepatic toxemia of pregnancy. Slemons found that normal values prevailed in preëclamptic toxemias, but convulsions caused a rise in the blood sugar. High arterial tension is given as a probable cause of hyperglycemia. This, however, was not a factor in cases of vomiting.

Hyperglycemia in the postpartum period is probably due to metabolic changes associated with the general process of involution; the endocrine system shares in this to an unknown degree. In early pregnancy, metabolism is so markedly changed that the reaction of the patient to the new metabolism may or may not produce toxemia. In cases in which toxic symptoms develop an increase in the blood sugar has been noted. Repetition of these observations with repeated basal metabolic readings and more frequent postpartum blood examinations would be of great value.

SUMMARY

1. The average range for blood sugar concentration in normal pregnant women is 0.09 per cent to 0.11 per cent which is the same as that found in nonpregnant women.

2. The average value in fifty-three observations of the blood sugar concentration during pregnancy was 0.11 per cent.

3. The average value in thirty-two observations of the sugar concentration of fetal blood taken from the umbilical cord immediately after delivery was 0.09 per cent.

4. The average value for twenty-two observations of the blood sugar concentration on the second day postpartum was 0.14 per cent.

5. Placental interchange of glucose is undoubtedly dependent on the higher concentration of blood sugar in the mother.

6. The influence of muscular exertion during labor is not a factor in postpartum hyperglycemia.

7. Anesthesia with ether is a contributing but not a determining factor in producing a rise in the sugar concentration of the cord blood.

8. Asphyxia produces a more marked rise than anesthesia with ether.

9. Involution of the uterus cannot be shown to be the primary factor in producing postpartum hyperglycemia.

10. The general physiologic change associated with involution may be a factor in producing postpartum hyperglycemia.

11. In certain types of toxemia there is an increase in the blood sugar concentration.

BIBLIOGRAPHY

(1) *Benthin, W.:* Ztschr. f. Geburtsh u. Gynäk., 1911, lxix, 198-212. (2) *Bergsma, E.:* Ztschr. f. Geburtsh u. Gynäk., 1912, lxxii, 105-153. (3) *Caldwell, W. E., and Lyle, W. G.:* Am. Jour. Obst. and Gynec., 1921, ii, 17-34. (4) *Frank, E.:* Ztschr. f. physiol. Chem., 1910-1911, lxx, 291-299. (5) *Guy, R. A.:* Quart. Jour. Med., 1921, xv, 9-17. (6) *Hirsch, E. F.:* Proc. Inst. Med., 1921, iii, 192. (7) *Kampf:* Quoted by Morriss, W. H. (8) *Killian, J. A., and Sherwin, C. P.:* Am. Jour. Obst. and Gynec., 1921, ii, 6-16. (9) *Kingsbury, F. B., and Sedgwick, J. P.:* Jour. Biol. Chem., 1917, xxxi, 261-268. (10) *Langfeldt, E.:* Jour. Biol. Chem., 1921, xlvi, 381-390. (11) *Lewis, R. C., and Benedict, S. R.:* Jour. Biol Chem., 1915, xx, 61-72. (12) *McGuigan, H., and Ross, E. L.:* Am. Jour. Physiol., 1915-1916, xxxix, 480-490. (13) *Morriss, W. H.:* Bull. Johns Hopkins Hosp., 1917, xxviii, 140-146. (14) *Neubauer, E., and Novak, J.:* Deutsch. med. Wchnschr., 1911, xxvii, 2287-2289. (15) *Schiller, H.:* Surg., Gynec. and Obst., 1919, xxix, 450-454. (16) *Schirokauer, H.:* Berl. klin. Wchnschr., 1912, xlix, 500-503. (17) *Scott, E. L. and Hastings, A. B.:* Proc. Soc. Exp. Biol. and Med., 1920, xvii, 120-122. (18) *Sedgwick, J. P., and Ziegler, M. R.:* Am. Jour. Dis. Child., 1920, xix, 429-432. (19) *Slemons, J. M.:* Analysis of the Blood in Eclampsia and Allied Intoxications. Am. Jour. Obst., 1918, lxxvii, 797-806. (20) *Slemons, J. M., and Morriss, W. H.:* Bull. Johns Hopkins Hosp., 1916, xxvii, 343-350. (21) *Stewart, G. N., and Rogoff, J. M.:* Am. Jour. Physiol., 1920-1921, li, 366-377.

SUGAR TEST IN PREGNANCY

By W. E. Welz, M.D., F.A.C.S., and A. E. Van Nest, M.D.,
Detroit, Mich.

From the Prenatal Clinic of the Department of Health, Detroit

FOR many years, some definite sign or aid in the early diagnosis of pregnancy (within first three months) has been sought. Abderhalden's use of ferments for digestion of placental protein for a diagnosis of pregnancy has not been of value to the desired degree; also the decrease in precipitation of the red blood corpuscles is not a specific criterion of pregnancy but is present wherever protein is being broken down.

It has long been noted that glycosuria in pregnancy is a frequent occurrence. In 1914 Franz Jäger,[1] working under Döderlein, used 100 gms. of levulose as a test for liver function in pregnancy, and found it present in the urine in 65 per cent of his cases, which was interpreted as a sign of insufficiency of the liver in pregnancy.

In December, 1920, Frank and Nothmann,[2] as a result of their investigations, reported that in thirty pregnant women alimentary glycosuria was produced in every case during the first three months of pregnancy, showing that renal glycosuria thus produced can be used as a means of early diagnosis of pregnancy, when other tests can furnish no definite diagnosis.

In September, 1921, Ludwig Nürnburger[3] also reported seventy-one cases in which it was shown that where there was a pregnancy of the first trimester, the sugar reaction was positive; and, in those cases where no pregnancy existed, a negative result was obtained.

If a normal person is fed 100 to 150 grams of grape sugar, the blood sugar increases normally 0.07 per cent to 0.14 per cent without the appearance of sugar in the urine. Frank has shown in normal persons that a hyperglycemia up to 0.19 per cent is not normally accompanied by the excretion of sugar by the kidneys, but with pregnant women an alimentary administration of even 100 grams of grape sugar leads to glycosuria with blood sugar under 0.19 per cent.

But here the blood sugar does not exceed the boundaries of physiologic hyperglycemia. On the basis of numerous researches, it may now be said that the spontaneous and artificial glycosuria of pregnancy occurs without an important increase of blood sugar content. Hence this renal glycosuria of early pregnancy may be used to diagnose pregnancy during the first trimester. Differentiation from other glycosurias, which are accompanied by hyperglycemia, such as diabetes mellitus,

primary liver disease, and the hypersecretions of internal glands, pituitary, thyroid, suprarenal, can be made by the low blood sugar percentage in pregnancy glycosuria.

This artificial renal glycosuria is present constantly in pregnancy only during the first three months, when other signs of pregnancy are inconclusive. After the twelfth week of pregnancy, it rapidly disappears, and can be evoked in the later months only in a definite percentage of cases (about 30 per cent).

We have used this method of diagnosis of early pregnancy in 71 cases. In the first 27 cases 100 grams of glucose were used. As this was not entirely satisfactory, a change to 150 grams was made. As the cases reported from Germany were from women accustomed to a low sugar intake, and as American women are accustomed to a high daily intake it was thought that a larger amount of sugar could be used with advantage in our cases. Folin and Berglund[4] have recently shown that 200 grams of glucose can be taken at one time without producing any glycuresis.

METHOD OF TEST

The patient on first being seen is told to come to the clinic the next morning without breakfast. The patient is then catheterized, the urine examined; and, if normal, the test is continued, and a specimen of blood taken for blood chemistry. Then we give 150 grams glucose dissolved in 500 c.c. of tea. The patient is required to keep the prone position for forty-five minutes. If the patient is allowed up and around, vomiting very often ensues during this period.

Forty-five minutes after sugar is taken, the bladder is catheterized, and the urine examined for sugar; again at one hour and one and one-half hours, the urine is taken and examined. Immediately after finding the sugar in the urine, a second blood specimen is taken for an estimation of blood sugar. This blood for second sugar estimation is usually taken between the one hour and one and one-half hour periods.

Of our first series of tests (27) using 100 gms. glucose, seven were at periods ranging from 14 to 24 weeks pregnant. As these were all advanced too far in pregnancy to diagnose by the sugar test, they may be ruled out entirely. Only one of these seven gave a positive result, that at 14 weeks; two had excessive hyperglycemia, 307-410 mg. per 100 c.c. of blood and were not considered as diagnostic of pregnancy. Of the other 18, which were under 12 weeks' period, fifteen agreed with the final diagnosis and three disagreed. In this series the urine was examined only at 45 minutes and 1 hour.

Our second series of cases using 150 grams of glucose and catheterizing at 45 minutes, one hour and one and one-half hours are as shown in Table I.

TABLE I

	DATE	SUGAR TEST	LAST MENSES	FINAL DIAGNOSIS	ESTIMATED PHYSICALLY TIME OF TEST	BLOOD SUGAR
1	1-17-22	Neg.	11- 1-21	Pregnant	17 Weeks	142.9 mg.
2	1-17-22	Pos.	11-10-21	Pregnant	10 Weeks	113.9
3	1-19-22	Pos.	11-17-21	Pregnant	8 Weeks	144.9
4	1-19-22	Neg.	11- 1-21	Not Pregnant		216.2
5	1-21-22	Pos.	11-15-21	Pregnant	9 Weeks	229.9
6	1-23-22	Pos.	11- ?-21	Pregnant	8 Weeks	125
7	1-23-22	Pos.	9-17-21	Pregnant	15 Weeks	171.6
8	1-28-22	Neg.	11-28-21	Not Pregnant		140
9	2- 2-22	Pos.	12-21-21	Pregnant	6 Weeks	254.8
10	2- 9-22	Pos.	12-10-21	Pregnant	8 Weeks	137.9
11	2- 9-22	Pos.	12- 8-21	Pregnant	9 Weeks	168
12	2-11-22	Neg.	12-23-21	Not Pregnant		150
13	2-16-22	Pos.	11-14-21	Pregnant	13 Weeks	183.5
14	2-16-22	Pos.	12- 8-21	Pregnant	9 Weeks	114.9
15	2-18-22	Neg.	2-16-22	Not Pregnant		190.1
16	3- 4-22	Pos.	1-14-22	Pregnant	7 Weeks	181.8
17	3- 4-22	Pos.	1-29-22	Pregnant	5 Weeks	209.4
18	3- 8-22	Pos.	12- 8-22	Pregnant	9 Weeks	213.9
19	3- 8-22	Pos.	11-30-21	Pregnant	15 Weeks	258.1
20	3-10-22	Pos.	1-31-22	Pregnant	5 Weeks	139.1
21	3-20-22	Neg.	12-15-22	Pregnant	15 Weeks	132
22	3-20-22	Pos.	12-20-22	Pregnant	13 Weeks	176.5
23	3-25-22	Neg.	12-20-22	Pregnant	14 Weeks	148
24	3-30-22	Pos.	3-12-22	Pregnant	10 Weeks	164.6
25	4- 6-22	Neg.	3- 1-22	Not Pregnant		135.8
26	4- 8-22	Neg.	1- 8-22	Pregnant	16 Weeks	88.5
27	4- 8-22	Neg.	1-10-22	Not Pregnant		141.9
28	4-13-22	Pos.	2- 3-22	Pregnant	12 Weeks	130.1
29	4-20-22	Neg.	4- 3-22	Not Pregnant		148.8
30	4-20-22	Neg.	11-28-22	Pregnant	16 Weeks	139.8
31	4-27-22	Neg.	2-25-22	Pregnant	8 Weeks	107.5
32	5- 6-22	Neg.	3- 6-22	Not Pregnant	8 Weeks	144.2
33	5- 6-22	Pos.	4- 6-22	Pregnant	12 Weeks	95.1
34	5-11-22	Neg.	3- 1-22	Pregnant	9 Weeks	148.8
35	5-11-22	Pos.	2-22-22	Pregnant	11 Weeks	151.2
36	5-13-22	Neg.	2-25-22	Pregnant	20 Weeks	161.8
37	5-18-22	Pos.	4-30-22	Pregnant	8 Weeks	186
38	5-18-22	Neg.	3-29-22	Not Pregnant	6 Weeks	131.8
39	5-25-22	Neg.	3- ?-22	Not Pregnant	6-8 Weeks	144.2
40	5-29-22	Pos.	3-25-22	Pregnant	8 Weeks	140.8
41	6- 1-22	Pos.	3-21-22	Pregnant	11-12 Weeks	156.3
42	6- 3-22	Neg.	4- 7-22	Not Pregnant		Spoiled
43	6- 8-22	Pos.	5-? -22	Pregnant	6 Weeks	85.1
44	6-10-22	Pos.	4- 2-22	Pregnant	12 Weeks	181.8

It will be seen from the above 44 cases only two cases (31 and 34) of those within the twelve weeks of pregnancy disagreed. These failed to give a positive sugar test, though later proved to be pregnant. In no case have we had a positive glycosuria, where there was no pregnancy, though having blood sugar estimations as high as 216.2 mg. per 100 c.c. of blood (Case 4). It will also be noted that Cases 4, 5, 17, 18 and 19, though agreeing with the final diagnosis, were of no value as there was a definite hyperglycemia present, which could be due to any one of the before-mentioned diseases (diabetes mellitus, primary liver disease, internal gland disturbance), but after

careful examination we are of the opinion that these were all simple alimentary hyperglycemias.

Blood sugars were determined by the method of Folin, urine sugars by ordinary Benedict's and Fehling's solutions. The percentage of sugar in the urine was usually between 0.2 and 1.5 per cent. Though most of the positive cases will give a positive urine at 45 minutes, some will not show positive until the one hour or hour and a half sample of urine. Since adding the one hour and a half examination, we have found four which did not give a positive reaction until the last specimen was examined.

Nürnburger and his coworkers claim that in abortions of the first three months, the sugar reaction remains positive as long as the placenta remains attached, for the greatest part, to the uterine wall. We have not been able to follow our cases this far, as our work is in a prenatal clinic, but we have had three cases, in which there were signs of threatened abortion, with two positive and one negative results; all three aborted within ten days. Both Frank and Nothmann and Nürnburger found the sugar test positive in all their cases of extrauterine pregnancy also. We have not so far had occasion to test such a patient.

Whether this glycosuria of early pregnancy is the result of liver insufficiency as Döderlein has shown to exist in practically all cases, or whether there is a definitely increased permeability of the kidneys to glucose during this period, is probably still debatable. But from the recent work of Frank in Germany, who claims that 0.19 per cent blood sugar is the renal threshold for glucose in the normal person, and also the work in this country of Folin and Berglund, who have come to the conclusion that the usual renal threshold is between 0.16 per cent and 0.18 per cent, we are inclined to believe that there is a definite lowered threshold of kidneys for glucose during the first twelve weeks of pregnancy. On the other hand, Nürnburger assumed that the calcium ions exerted a colloid-fast effect on the kidney cells, and thereby accomplished a thickening of the kidney filter, but in spite of the administration of relatively large doses of calcium, he was not successful in influencing the renal glycosuria of pregnancy.

We believe that a spontaneous or artificially induced renal glycosuria, with a blood sugar estimation below 0.19 per cent in the first 12 weeks after conception, is a valuable aid in the early diagnosis of pregnancy. In our experience the test has proved correct in more than 95 per cent of our cases.

REFERENCES

(1) Ztschr. f. Geburtsh. u. Gynäk., lxxiv. (2) München. med. Wchnschr., Dec. 10, 1920. (3) Deutsch. med. Wchnschr., Sept. 22, 1921. (4) Jour. Biol. Chem., li, No. 1.

THE PATHOLOGY OF UTERINE BLEEDING IN 100 ANALYZED CASES*

By Herman Grad, M.D., F.A.C.S., New York City, N. Y.

BLEEDING from the uterus, aside from that of menstruation, occurs together with a great number of pathologic lesions of the female pelvic organs. Occurring under these varied conditions of pelvic pathology, it is difficult to diagnose the cause of the bleeding in every case.

For the study of uterine bleeding the histories of 100 cases of this affection were taken from the records of the Woman's Hospital. These histories and pathologic findings were carefully studied and made the basis of this paper. The clinical investigation of the cases convinced me early in the study that uterine bleeding must be classified under six different groups, as in each group there is a definite etiological factor causing the bleeding. On the basis of these etiological factors the classification was made and the six groups of cases so classified are shown in Chart I.

Clinically every case of uterine bleeding can be classified under one of the above groups. It is obvious, however, that some cases of uterine bleeding may have one or more causative factors given in Chart I active at the same time. For example, a patient may suffer with an infection, may have a neoplasm in the uterus and be harassed with endocrine distress, or a pathologic gestation may be present coexistent with a neoplasm and infection. Under these complex pathologic conditions it would indeed be difficult to state the precise cause of the uterine bleeding. This is a very important fact to consider, and the study of these 100 cases of uterine bleeding brings out this fact very forcibly. The cases of uterine bleeding should be grouped according to what the clinician considers the most prominent symptoms in the case, and what he considers the most prominent, etiologic factor.

When a case of persistent uterine bleeding presents itself, before deciding on the therapeutic measures for relief, an endeavor should be made to place the case into one of the six groups enumerated above. By doing this the clinician will have made a very important step toward giving proper therapeutic measures for the relief and cure of the case in question.

The study of the 100 cases of uterine bleeding has brought to light

*Read at a meeting of the New York Obstetrical Society, May 9, 1922.

many interesting facts. I shall take up each group of cases by itself and point out the cause of the bleeding.

Pregnancy.—In the first group where gestation was the etiologic factor in the uterine bleeding, eight out of thirteen had retained secundines, the removal of which by curettage brought prompt relief.

Several interesting facts came to light by the study of this group, which I shall give here in detail, as I consider these facts of some importance.

In two cases, Nos. 7 and 36, an early abortion occurred five weeks before the cases came under observation. The patients complained of a

CHART No 1.
100 CASES OF UTERINE BLEEDING
Classified in 6 Groups

Group	Etiology	Pathology	Number of Cases
1	Pregnancy	Abortion. Uterine Mole. Hydatidiform Mole. Chorio-Epithelioma. Diseases of Placenta. Pathologic Implantation of Ovum. Ectopic Gestation. Malposition of Placenta.	13
2	Infection	Endometritis. Metritis. Salpingitis. Oophoritis.	23
3	Neoplasms of Uterus & Ovaries.	Polypoid Conditions of Uterus and Cervix. Fibroids. Carcinoma and Sarcoma Polyps. Ovarian Neoplasms.	38
4	Displacements Lacerations Congestion.	Retroversion. Inversion of Uterus. Subinvolution. Lacerations. Prolapsus Uteri.	13
5	Endocrine Distress	Hyper-function of Ovaries Hypo-thyroidism Disease of Hypophysis and Adrenals etc. Disturbed Balance of Function of Ductless Glands.	9
6	Constitutional Causes and Blood Diskrasie	Cardio-Vascular Disease Nephritis. Phthisis. Infectious Diseases, Rheumatoid Conditions Anaemia. Chlorosis. Pernicious Anaemia.	2
		Records incomplete	2
		Total	100

metrorrhagia of five weeks' standing. Pelvic examination was negative. Curettage brought away a very small piece of retained secundines in both cases and the rest of the curettings showed as the pathologist expressed it, "typical premenstrual mucosa." These cases bring out the fact that after abortion, the uterine mucosa regenerates promptly preparatory for the normal menstrual cycle of the coming month, in spite of the fact that the uterine cavity harbored a small portion of retained secundi.

No. 29 was also an interesting case in this group. The patient was forty-three years old and had three children. Examination showed a fibroid of the uterus, a cervical polyp and there was a history of an abortion. On account of the abortion the case was placed in the first group instead of the third, under neoplasm. A curettage resulted in a cure of the uterine bleeding of five weeks' standing. The pathologist made the following report: "decidua compacta and spongiosa, adenoma

polyposum of the cervix. Polyp shows large cervical glands of the hyperplastic type." The cause of the uterine bleeding was retained secundines, the polyp and fibroid having nothing to do with the bleeding.

In this group of uterine bleeding there were three cases due to ectopic gestation, Nos. 4, 9, and 58. The pathologist shows two inter-

Uterine Bleeding

CHART No II

PREGNANCY, 13 CASES

Pathologic Report

No	Endometrium	Uterine Glands	Uterine Tubes	Ovaries	Duration of Uterine Bleed	Age	No. of Pregnancy	Operation
4	Not stated	Not stated	Ectopic	Normal	4 Wks	27	2	D&C. Trachelorrhaphy Left Salpingectomy-Ectopic Vent. Susp. App.
7	Premenstrual History of Abortion	Premenstr'l	Normal	"	2 Months	25	1	Divulsion & Curettage
9	Not stated	Not stated	Ectopic in stump of Tube	"	1 Year	31	None	Removal of stump of tubes previously resected
19	Placental tissue	"	Normal	"	1 Month	29	3	Spontaneous expulsion of Foetus & Placenta
23	Low impl'td Placenta	"	"	"	3 "	20	1	No Operation Pregnancy
25	Retained secundinae		"	"	1 "	30	3	Divulsion & Curettage
29	Dec. comp.& Spong. ordrm.pain	Hyperplastic glands of Cervix	"	"	1 "	43	3	Removal of polyp. Divulsion & Curettage
36	Premenstrual History of Abortion	Premenstrual	"	"	6 Wks	32	4	Divulsion & Curettage
40	Chorio-Epithelioma	Not stated	"	"	"	27	None	Pan-Hystero bil. salpingo-oopherectomy
42	Retained secundi.		"	"	3 Months	29	?	Divulsion & Curettage
50	Normal	Normal	"	"	6 Wks	39	7	Spontan's expulsion of Foetus & Placenta
58	Round cell Infiltration	Distorted Glands	Ectopic	"	2 Months	29	2	D&C Repair of Ant. Vag. Wall. Bil. Salpingectomy
100	Early Placenta	Not stated	Normal	"	6 Wks	29	4	D&C Trachelorrhaphy

Summary of Causes of Uterine Bleeding in 13 Cases

4 Causes

	CASES	
1	8	Abortion Nos 7-19-25-29-36-42-50-100
2	3	Ectopic Gestation 4-9-58
3	1	Low Implanted Placenta 23
4	1	Chorio-epithelioma 40

esting facts in the specimens. In Case No. 9, the gestation occurred in the remnant of the fallopian tube, that had been resected at a previous operation. Ectopic gestation in stumps of resected tubes has been observed repeatedly. It seems to me therefore, in the light of these repeated observations, that resection of the uterine tubes is a very questionable procedure. It is a question whether a resected tube is ever a functionating organ. A resected tube appears to be more of a menace

to the individual than a benefit. In this case the patient was thirty-one years old. She had one child. She was laparotomized for chronic salpingitis and had the right tube resected. Four years later an ectopic gestation occurred in the resected tube. An interesting question arises, in the light of these observations, how does the fecundated ovum reach the remnant of a resected fallopian tube? I have, as others have, repeatedly observed in reopened abdomens, that the resected tube was firmly adherent to the broad ligament and no osteum existed. Therefore, if such are the facts, a fecundated ovum can only reach this remnant of the tube by way of its open uterine end, by migration through the opposite tube. The pathologist says: "section of the tube stump shows early pregnancy, with double layer of epithelioma on surface." This shows conclusively that the tube was adherent, and one would hardly expect to find an open distal end of a tube so adherent.

In the second case, No. 58, the patient was twenty-nine years old and had two children. She had a metrorrhagia of two month's duration. She had the following operations: divulsion and curettage, repair of anterior wall, bilateral salpingectomy, ectopic on right side. The pathologist makes the following report: "Chronic endometritis. Tubal gestation. Tube contains blood clots. Section of tube shows fairly well preserved chorionic villi. Section of curettings shows remnants of mucosa with numerous round cells in the stroma and a few distorted glands."

This case of ectopic gestation is here reported because a bilateral salpingectomy was done. This brings up an important question, namely, under what conditions should the opposite fallopian tube be removed in operating for ectopic gestation? It is in the experience of every gynecologist that in ectopic gestation the fallopian tube opposite to the gravid one, looks congested, thickened and engorged. It is very difficult to say if the tube is diseased or normal. It is very frequently a difficult problem to decide, while the abdomen is open, what it is best to do. Is there anything that can guide one in deciding what best to do to avoid the mistake of removing a normal tube or leaving behind a diseased one?

On two very important occasions this question confronted me. The tube opposite to the ectopic looked diseased, with a gonorrheal history, behind the ectopic. I decided against a salpingectomy and I am glad to say that in one of these cases, since the operation, two children were born, while in the second case no symptoms of pyosalpinx developed so far although no pregnancy has occurred. In both of these cases the removal of the tubes was seriously contemplated at the time of operation and I refrained from doing so only on account of the youth of the patients. I should advise against salpingectomy of the opposite tube in ectopic gestation in the light of my own experience, and should

remove the opposite tube only in those cases where the tube is so hopelessly diseased that it is beyond the possibility of natural repair.

What is the cause of the uterine bleeding in this group of cases? In the abortion cases the uterine bleeding is readily understood. Tissue changes and cell destruction result in the escape of blood and hence the uterine bleeding. In the early months of pregnancy before the placenta forms, faulty implantation results in tissue destruction and a necessary escape of blood. After the placenta has formed bleeding may occur from disease of the placenta or faulty position, or the placenta may undergo changes like in the chorioepithelioma. In tubal gestation the bleeding may occur from two sources. First from the gestation sac itself as a result of faulty implantation, the blood reaching the uterine cavity by way of the patent uterine end of the tube, or secondly the uterine bleeding may have its source from the endometrium, a proliferation having taken place in the endometrium as a result of the tubal gestation.

Infection.—In the second group of cases where infection was the etiologic factor there were 23 cases. A noteworthy feature in this group is the fact, that in every specimen subjected to pathologic examination the endometrium showed pathologic changes. Chart III shows these pathologic changes in detail in the 23 cases belonging to this group. In four cases out of 23 a polypoid condition of the endometrium was encountered. In 11 cases out of 23 chronic endometritis was found. In two cases the endometrium was found edematous, thick and pale. In one case the pathologist reports that the endometrium showed various phases of menstruation. The fallopian tubes had round-cell infiltration in four cases. Hydrosalpinx was present in two cases, pyosalpinx three times and in 11 cases the tubes were found normal. The ovaries showed cystic degeneration in four cases. In 18 cases the ovaries were normal. In one case the ovaries were carcinomatous.

From this summary it is very obvious that in cases with menstrual disturbance and uterine bleeding where infection is the etiology, 20 per cent show a polypoid condition of the endometrium; 55 per cent an inflammatory condition of the endometrium with round-cell infiltration and the remaining 25 per cent have various other pathologic disturbances. Endometrial pathology is evidently the most frequent cause of uterine bleeding in infection of the female generative organs. The onslaught of the infection is borne by the endometrium which has to bear the brunt of the attack of the microorganisms, and although the uterine glands and endometrium undergo a physiologic regeneration from month to month, the endometrium cannot entirely throw off the invasion of the microorganisms and get rid of the inflammatory exudate, which is present in the form of a round-cell infiltration in over 55 per cent of cases.

Uterine Bleeding

CHART No III-a

INFECTION 23 CASES

Pathologic Report

No	Endometrium	Uterine Glands	Uterine Tubes	Ovaries	Duration of Uterine Bleeding	Age	No. of Pregn's	Operation
3	Endometritis, Thick polypoid growth, confluent mucous formation	Interval type	Normal	Normal	4 Months	39	2	Pan-Hysterectomy Bil Salpingo-oophorectomy
6	Adenoma polyposum	Hyperplastic	Rd cell infiltration	Carcinoma	2 years	50	3	Pan-Hysterectomy Bil Salpingo-oophorectomy
12	Myoma polyposum	Premenstrual	Hydrosalpinx	Normal	3 Months	45	0	Supr. vag. Hysterect. Bil Salpingo-oophorect
15	Round cell infiltration	Interval type	Inflammation Rd cell infiltration	Pol cysts	2 years	21	0	Supr vag Hysterect Bil Salpingo-oophorect
18	Various phases of menstruation	Various phases of menstruation	Pyosalpinx	Pol cysts	2 Weeks	46	1	Hysterectomy Bil salp.-oophorect
27	Oedematous, thick and pale	No change	Granulation tissue	Normal	5 years	44	1	Hysterectomy
30	Ch. Hemorrhagic Endometritis	Premenstrual changes	Normal	"	7 "	32	3	Pan-hysterectomy Left salpingo-oophorectomy
31	Oedema	Hyperplasia	"	"	6 Months	39	2	Pan-hysterectomy Bil Salp-oophorect
33	Thickened Blsp. Rd cell infiltration Hyalin degeneration	"	"	"	2 years	46	7	Pan-hysterect Bil. Salp.-oophorect
39	Ch. Endometritis Rd cell infiltration	"	"	"	1 Month	26	1	D&C cured
45	" Ch. Endometritis	Hyperplastic Rd. cell infiltrat'n	"	"	6 Weeks	30	0	D&C
47	Ch. Endometritis	"	"	"	5 "	21	1	D&C Pessary
48	Round cell infiltration Hyperplastic	Hyperplastic	"	"	1 Year	41	3	D&C Good result
51	Not observed	Not observed	Thickened Rd cell infiltrat'n	"	6 Months	31	3	Bil salpingectomy Good result
53	Ch. Endometritis Rd. cell infiltration Hyaline degeneration of vessel walls	"	Normal		6 "	32	2	Vag. hysterect Left Salp.-oophorect
54	Not observed	"	Diseased Ch. pen salpingitis	"	4 "	28	0	Left salpingectomy Short'g round lig. Appendix
61	Not observed	"	Sub. acute Salp. Rd cell infiltration	"	5 years	23	0	Bil salpingectomy
65	Not stated	Not stated	Buried in adhesions	Buried in adhesions	11 "	30	2	Freeing of adhesions of adnexae Removal of cyst Hydatid of Morgagni
76	Ch. Endometritis Round cell infiltration	"	Normal	Normal	6 Months	28	0	D&C & Radium
83	Hyaline degeneration	Hyperplastic	Hydrosalpinx	Pol cysts	Many years	49	4	Hysterectomy Bil Salp.-oophorect
86	Ch. Endometritis Rd. cell infiltration	"	Pyosalpinx	"	3 Weeks	19	0	Hysterectomy Bil Salp.-oophorect
90	Ch. Endometritis Rd cell infiltration Oedematous	Distorted Hyperplastic	Normal	Normal	4 "	21	1	D & C Good result no bleeding
99	Not observed	Not observed	Pyosalpinx	Hemorr. Corpus luteum	6 "	29	4	Bil salpingectomy Rt. oophorectomy Myomectomy

CHART No III-b

Summary of Causes of Uterine Bleeding in Cases of Infection

2 Causes

1	55%	11	Endometritis Nos 15-30-39-45-47-48-53-76-83-86-90
2	19%	4	Polypoid Condition of Endometrium Nos 3-6-12-33
		1	Various Phases of Menstruation No 18
		2	Oedema of Endometrium Nos 27-31
		5	Not observed Nos 51-54-61-65-99
		23	

Uterine Tubes

45%	11	Normal Nos 3-30-31-33-39-45-47-48-53-76-90
	2	Hydro-salpinx Nos 12-83
	3	Pyo-salpinx Nos 18-86-99
	4	Round Cell Infiltration Nos 6-15-31-61
	3	Chronic Salpingitis Nos 54-65-27
	23	

Ovaries

80%	18	Normal Nos 3-12-27-30-31-33-39-45 47-48-51-53-54-61-65-76-86-90
	4	Cystic Nos 15-18-83-90
	1	Carcinoma No 6
	23	

That this is a correct interpretation of the pathologic findings in these cases is further borne out by the study of the cases tabulated in Chart III. In 45 per cent of the cases of uterine bleeding the uterine

tubes were found normal and the ovaries were normal in 80 per cent of the cases. In Case No. 3, the patient 39 years old, who had two children, was suffering with a metrorrhagia of four months' duration. A panhysterectomy and a double salpingooophorectomy was done. When the specimen was examined both tubes and both ovaries were found normal. The endometrium was found diseased and presented a thick polypoid growth, which was edematous and had thrombosed vessels. In this case the metrorrhagia was due to the polypoid growth in the endometrium, supplemented or perhaps aggravated by the endometritis. Again in Case 30, a woman 32 years old, with a history of metrorrhagia off and on for seven years, was operated on, and the specimen showed normal tubes and ovaries, with a diseased endometrium to account for the repeated attacks of metrorrhagia of many years' standing.

Taking these facts into consideration one is justified in concluding that uterine bleeding in cases of infection is due to pathologic changes in the endometrium in the form of an infection, and that a certain number of cases of uterine bleeding are due to a polypoid condition of the endometrium, which occurs in at least 20 per cent of cases. This brings up an important question, namely, that of curettage for metrorrhagia in cases with a history of infection. The teaching has been that curettage should not be practiced in cases of infection. This is a very sane teaching, as during curettage the protective leucocytic layer which acts as a bar to the further spread of infection, is removed by the curette. Nevertheless, in view of the fact that nearly every endometrium is diseased in cases of metrorrhagia with history of infection, a curettage should be performed in every case of metrorrhagia where the operator does not contemplate the removal of the uterus.

Cases 39 and 61 bear upon this subject and bring out another interesting point in reference to curettage. In Case 39 a young woman 26 years old, with one child, was suffering with a severe metrorrhagia of one month's duration. Her uterine adnexae were apparently normal. She was curetted and the specimen showed endometritis with round-cell infiltration. Subsequent history showed that she was relieved of all symptoms. In contrast to this case of beneficial curettage is Case 61.

She is also a young woman twenty-three years old, who has been suffering with repeated attacks of metrorrhagia for five years. She was laparotomized and a bilateral salpingectomy done. The specimen showed subacute salpingitis and round-cell infiltration. She was not curetted and while she is relieved of the symptoms of salpingitis her metrorrhagia did not improve and a curettage is indicated for relief of symptoms. A curettage at the time of operation would have been the proper procedure. In these cases the sharp curette, which is the instrument to be used, removes pathologic tissue debris, and in that way benefits the condition. Whether the curettage is of temporary or of

permanent benefit is another question. There is, however, no doubt that the temporary benefit of the curettage is certain and definite.

Neoplasms.—Under the third group, there were 38 cases, tabulated in Chart IV. The study of this chart shows that there were eight cases of cancer of cervix; 26 cases of fibroids and four cases with uterine polyps. The endometrium was affected in nearly every one of the 38 cases. What was the cause of the uterine bleeding in these cases? In eight there was the cell destruction which is present in all carcinomas. In Case 72 there was a history of uterine bleeding for a year, in a

Uterine Bleeding

CHART No IV-a

NEOPLASMS 38 CASES

Pathologic Report

No	Uterus	Cervix	Endometrium	Uterine Glands	Uterine Tubes	Ovaries	Duration of Metrorrhagia	Age	Number of Pregnan'c's	Operation
1	Normal	Carcinoma	Hyperplastic	Hyperplastic	Normal	Normal	4 Months	62	2	Dk. Radium 100 mg. 24 hrs.
5	Carcinoma	Carcinoma	Carcinoma	Carcinoma	"	"	3 Months	58	None	Radium Application
8	"	"	Atrophic	Atrophic	"	"	1 "	40	1	" "
10	"	"	Carcinoma	Carcinoma	"	"	3 "	31	None	" "
14	"	"	"	"	"	"	3 "	36	4	" "
17	Fibroid	Normal	Adeno-myometritis	Penetrate Musculatis	"	Fol. cysts	3 years	33	None	Supra-vag. Hysterectomy Rt Salpingo-oophorectomy
20	Adeno-myoma	"	No inflammatory changes	Penetrate Musculature	"	"	3 "	42	"	Supra vag Hysterectomy Bil. salpingo-oophorectomy
21	Polyp at Fundus	"	Round cell Infiltration	Not stated	"	"	3 Months	40	"	Pan. hysterectomy Left Salpingo-oophorectomy
22	Several Fibroids	"	Thickened	Premenstrual changes	Diseased	"	2 "	44	2	Supra-vag. Hysterectomy Bil. Salpingo-oophorectomy
24	Carcinoma	Carcinoma	Carcinoma	Not stated	Normal	"	2 "	42	2	Radium Application
32	Large Fibroma sub-mucous	Normal	Pale and hemorrhagic	" "	Hyperaemic	Hemorrhagic Cysts	6 "	56	4	Supr. vag Hysterectomy Bil. Salpingo-oophorectomy
35	Uterine Polyp	Polyp protrudes	Premenstrual Mucosa	Premenstrual	Normal	Normal	10 "	45	4	Removal of Polyp Radium Application
41	Fibroids	Normal	Oedema polyposum	Hyperplasia	"	Corpus luteum Cyst	1 year	Not stated	0	Supr.-vag. Hysterectomy Bil. Salpingo-oophorectomy
43	Carcinoma	Carcinoma	Carcinoma	Carcinoma	"	Normal	2 Months	56	1	Radium Application
44	Fibroids	Polyp protrudes	Interval	Interval	"	"	3 "	48	2	Removal of Polyp and Radium Application
46	Large Fibroids	Normal	Thickened	Premenstrual	Thickened Round cell infiltration	Large cyst Hemorrhagic	8 years	34	1	Supra-vag. Hysterectomy Bil. salp-oophorectomy
49	Fibroids	"	Hyperplasia	Hyperplasia	Normal	Corpus luteum Cyst Hemorrhagic	6 Months	38	None	Supra-vag. Hysterectomy Rt. salp-oophorectomy
56	Sub-mucous Fibroid	"	Thickened oedematous	Normal	"	Cyst	3 "	47	"	Supra-vag Hysterectomy Left salp-oophorectomy
62	Multiple sub-mucous Fibroids	"	Thin	Cystic Glands	"	Ov. Cyst	1 year	39	"	Supra-vag. Hystere-Bil. Salpingo-oophorectomy
66	Fibroids	"	Very thin destroyed by Iodin	Not stated	"	Normal	9 "	39	3	Pan-Hysterectomy Bil. salp-oophorectomy
67	"	"	Thin No other change	"	Hydro-salpinx Bilateral	"	2 "	43	None	Supra vag Hysterectomy Bil. salp-oophorectomy
68	"	"	Hemorrhagic Oedematous	Premenstrual	Normal	"	3 "	43	1	Supra-vag Hysterectomy Bil. salp-oophorectomy

woman 45 years old, the clinical diagnosis was carcinoma of the cervix, but the pathologist reported only necrotic tissue. The clinical diagnosis is the correct one. The pathologist did not receive a proper specimen for examination. Subtracting the eight cases of carcinoma, where the cause of bleeding is well known and readily understood, there are left 30 cases of fibroid of uterus, for the study of the cause of uterine bleeding.

Careful analysis shows that in the 30 cases of fibroids, the neoplasm was responsible for the bleeding in only 17 cases or in 55 per cent. In 11 cases out of the 30 the cause of the bleeding was infection of the

endometrium, the tumors having nothing to do with the bleeding. In other words, in 35 per cent of cases of fibroids of the uterus, the disturbance in menstruation and uterine bleeding is not caused by the neoplasm but is due to infection of the uterine endometrium, the cause of the bleeding being precisely the same as in cases of infection of the generative organs, namely diseased endometrium.

CHART No IV-b

No	Uterus	Cervix	Endometrium	Uterine Glands	Uterine Tubes	Ovaries	Duration of Metrorrhagia	Age	Number of Pregnan's	Operation
70	Large Tumor Leiomyoma uteri	Normal	Thickened and oedematous	Premenstrual	Hyperaemic	Cystic Ovary with Fibrosis of Stroma	2 Years	43	1	Supra-vag. Hysterect. Bil. salp.- oöphorect.
72	Normal	Carcinoma Infiltration	Normal	Not stated	"	"	1 "	45	4	D&C Radium
73	Multiple Fibroids	"	Very thin	Glands penetr. Myoma	Normal	Normal	6 Weeks	39	2	Supra-vag. Hysterect. Bil. salp.- oöphorect.
75	Fibroids	"	Not stated	Not stated	"	Corpus luteum cyst	3 "	32	0	Hysterotomy Append removal of tumor & cyst.
77	Degenerated with mural Fibroid	"	Normal	Normal	"	Normal	6 Years	30	Single	Supra-vag. Hysterect.
78	Adenoma Polyposum	Polyp protrudes	Hemorrhagic	Cystic	"	Serous Cyst	1½ "	?	"	" " " Bil. salp.- oöphorect.
79	Very large Fibroid	Normal	Very hyperaemic Adenoma polyposum	Not stated	"	Bicystic Large cysts	5 "	35	"	" " "
80	Sub-mucous Fibroids	"	Hemorrhagic No other change	"	"	Normal	2 "	34	0	Supra-vag. Hysterect.
81	Myoma uteri	"	Thin premenstrual	Premenstrual	"	"	6 "	31	"	" " " Left salp.- oöphorect.
84	Large Myoma	"	Adenoma polyposum	Not stated	"	Corpus luteum Cyst	8 Months	?	"	" " " Right " "
92	Large Fibroid	"	Thickened Round cell infiltr.	Penetrate Muscularis	Salpingitis Interstitialis	Serous Cyst	5 "	41	2	Pan Hysterec- double salp.- oöphorect.
93	Numerous Sub-mucous Fibroids	"	Hemorrhagic Areas	Not stated	Normal	Normal	3 Years	33	1	Supra-vag. Hysterect.
95	Large Polyp	Polyp protrudes	Not stated	"	"	"	2 "	42	3	Removal of Uterine polyp No bleeding later
96	Fibroids	Normal	Round cell infiltrate Hemorrhagic Foci	"	"	"	6 Months	52	1	Supra- vag. Hystero- Bil. salp.- oöphorect.
97	"	"	Oedematous	Hyperplastic	"	Small Retention Cyst	7 Years	36	Single	Supra-vag. Hysterect. Left salp.- oöphorect.
98	Sub-mucous Myoma	"	Oedematous Hemorrhagic	Not stated	"	Normal	7 " part very much	42	1	" " " Bil. salp.- oöphorect.

Summary of the Causes of Uterine Bleeding in Cases of Neoplasms

Seven Causes

	Cases			
1	8	Carcinoma Nos 1-5-8-10-14-24-43-72	21%	Condition of Uterine Adnexa
2	7	Adenoma polyposum of Uterus or Cervix 35-41-44-78-79-84-95	20%	Uterine Tubes: Normal 32 / Diseased 6 / Total 38 — Ovaries: Normal 25 / Diseased 13 / Total 38
3	11	Endometritis 21-22-46-66-67-68-70-81-92-96-97	30%	
4	6	Sub-mucous Fibroids 32-56-62-80-93-98	16%	
5	3	Penetrating uterine glands 17-20-73 (92 bis)	8%	
6	2	Hyperfunction of Ovaries 49-75		
7	1	Degeneration of Neoplasm 77		

In the 38 cases tabulated there were seven different causes encountered for the uterine bleeding, from a pathologic point of view. These seven different causes are as follows:

1. Cause—8 Carcinoma ..... 21 per cent
2. Cause—7 Adenoma polyposum of uterus ..... 20 per cent
3. Cause—11 Endometritis ..... 30 per cent
4. Cause—6 Submucous fibroids ..... 16 per cent
5. Cause—3 Penetrating uterine glands ..... 8 per cent
6. Cause—2 Ovarian hyperfunction
7. Cause—1 Degeneration of neoplasm and necrosis

From the study of these cases, I find that a polypoid condition of the endometrium appears to be a frequent cause of uterine bleeding. In Group II it was found that the above-mentioned condition was respon-

sible for uterine bleeding in 20 per cent of cases. The same is true of the third group of cases,—here again the condition was encountered by the pathologist in 20 per cent of cases or seven times.

What is this condition of the endometrium that is responsible for uterine bleeding in so large a number of cases? The pathologist calls the condition "adenoma polyposum uteri." It is a soft adenomatous structure of the endometrium containing large numbers of uterine glands and has a tendency to bleed. Ulceration in the form of a break in the surface epithelium occurs frequently on these polypoid structures and the loss of blood is from these areas of ulceration. These polypoid growths also suffer from edema and infection which is shown under the microscope by a round-cell infiltration. When polyps of larger size develop out of an endometrium so affected, the polyps protrude through the cervical canal and another mechanical factor is introduced which may cause uterine bleeding, namely, pressure necrosis of the polyp with cell destruction and escape of blood. Some of these polyps bleed very profusely and dangerous degrees of anemia may develop. Very frequently in necrosis of uterine polyps, an inflammatory reaction occurs in the myometrium and also in the uterine wall, introducing a dangerous factor, which may cause death from sepsis. From a surgical point of view these cases of necrosed polyps with sepsis are very poor surgical risks.

Polypoid condition of the endometrium is a pathologic entity that demands attention as an etiologic factor in uterine bleeding. It is the cause of menstrual disturbance and uterine bleeding in 20 per cent of all cases, whether the underlying factor is infection or neoplasm. I am inclined to believe that an invasion of the endometrium by microorganisms is a factor in the formation of this polypoid condition of the endometrium. It is, however, possible that endocrine disturbance may be an etiological factor. Be this as it may, adenoma polyposum uteri is a frequent cause of uterine bleeding and occurs in at least 20 per cent of all cases.

In 30 per cent of cases of this group of uterine bleeding with neoplasm, the cause of the bleeding must be ascribed to infection of the endometrium. We have here 11 cases in which the pathologist finds an inflammatory reaction in the endometrium and nothing else to account for the uterine bleeding. While these cases all had fibroids of the uterus, the uterine bleeding was due to a pathologic endometrium, the neoplasm playing but a secondary rôle in causing the hemorrhage.

In six cases of this group the specimens showed a submucous fibroid, which was responsible for the uterine bleeding. The specimens show that the uterine mucosa overlying these submucous tumors, undergoes a pathologic change. The mucosa becomes thin, the uterine glands

disappear or become fewer in number. In some cases the mucosa becomes fibrous and edematous and in others a hemorrhagic condition develops in the mucosa overlying the neoplasm. These pathologic conditions developed in 16 per cent of cases. It is a distinct pathologic entity and must be classed as one of the causes of uterine bleeding in fibroids of the uterus. The escape of blood takes place from the diseased mucosa. This pathologic change in the mucosa depends on the neoplasm directly. Whether this pathologic change in the mucosa is due to a pressure necrosis from the tumor or is a distinct disease by itself as a result of the neoplasm, is not known. The fact remains that in about 16 per cent of cases of uterine bleeding, submucous fibroids are found. The mucosa overlying these neoplasms becomes diseased and blood escapes from this damaged mucosa.

Another pathologic condition was encountered in these cases which is of interest. It was found that in three cases the uterine glands penetrated the musculature of the uterus to a very extensive degree. I shall speak of these three cases in detail, because it is important to determine whether this condition of penetrating, profuse and extensive proliferation of uterine glands, can be one of the causes of uterine bleeding in neoplasms of the uterus. From the study of these cases, I believe that extensive proliferation and penetration of uterine glands can readily be the cause of persistent uterine bleeding, because it is a disease of the uterine gland itself.

The history of these cases is as follows:

Case 20.—Patient 42 years of age, never pregnant, was suffering with metrorrhagia for three years. She had a good sized tumor in the uterus. A supravaginal hysterectomy and a bilateral salpingo-oophorectomy were done. When the specimen was examined, it was found that there was an adenomyoma of the uterus. The endometrium had no inflammatory changes. The tubes and ovaries were normal. The only pathologic change found in the specimen was an extensive proliferation of the corpus glands with deep penetration into the muscularis. In this case there is no other way to account for the severe metrorrhagia of three years' standing but to accept this pathologic change in the uterine glands as the cause of the uterine bleeding. In Case 17, the patient was 55 years old and had also a metrorrhagia of three years' standing. The specimen showed an adenomyometritis and penetrating uterine glands with no other abnormalities. In Case 73 we have the same condition. The patient was 39 years old, she had two children. There was a multiple fibroid of the uterus. After operation the specimen showed penetrating uterine glands with no other pathologic changes to account for the bleeding. In Case 92 the pathologist also reports penetrating uterine glands, but in this case, the endometrium also showed round-cell infiltration so it was not included among these cases. This case, however, is an additional proof and further shows that this condition of penetrating uterine glands and extensive proliferation, is a pathologic manifestation of uterine glands and is frequently encountered. It is something that should be taken into consideration, as a cause of uterine bleeding in neoplasm of the uterus.

In one case the pathologist noted that the uterine bleeding was due to degeneration of the tumor itself. It was in Case 77. The patient was a single woman, thirty years old, who was suffering with metrorrhagia for six years. A hysterectomy was done and the specimen showed a degenerated fibroid. The tumor itself was bleeding into the uterine cavity, as a result of cell destruction. While it is not advisable to theorize from one single case, and make deductions, it must be admitted that it is in the domain of great possibilities that a degeneration of a neoplasm occurs and can result in uterine bleeding. Be this as it may in this case here reported, no other deductions could be made. The uterine bleeding was caused by the breaking down of the neoplasm and therefore this condition of degeneration of the fibroid tumor itself is put down as one of the causes of uterine bleeding.

Uterine Bleeding
CHART No V.
DISPLACEMENT, CONGESTION AND LACERATIONS OF UTERUS 13 CASES
Pathologists Report

No	Uterus	Cervix	Endometrium	Uterine Glands	Perineum	Ant Vag Wall	Uterus Tubes	Ovaries	Duration of Bleeding	Age	No of Preg's	Operation	Results
26	Retroverted	Lacerated	Not stated	Not stated	Lacerated	Normal	Normal	Normal	2 Mths	28	2	Trachelorrhaphy Perineorrhaphy Per Abdomen	Wardell and normally since operation
37	Normal Gillian time	Repaired 4 mo before	Hyperplasia	Hyperplasia	Repaired 4 mo before	"	"	"	4 "	38	Single	D&C Rad 50mg 12hrs	Excellent result, passing normal after 6 months
55	Retroverted	Lacerated	Not stated	Not stated	Lacerated	Cystocele	"	"	2 Yrs	42	2	D&C Repair Cystocele Shortening Rd Lig Uterus Appendectomy	Excellent result
57	"	Lacerated No Cell Infiltration	"	"	Normal	Normal	"	"	3 Mths	27	1	D&C Amputation of Cervix Simpson operation	Excellent result
59	Normal	Lacerated	No material obtained by curettage		Lacerated	"	"	"	6 Wks	30	2	D&C Amputation of Cervix Repair of Peri Soon	Good result
71	"	"	Interval	Interval	"	"	"	"	2 Mths	41	?	D&C Amputation of Cervix Perineorrhaphy	Very good result No bleeding
74	Retroversion	Moderate Laceration	Not stated	Not stated	Moderately Lacerated	"	"	"	9 Wks	26	2	Shortening Round Ligament of Uterus	Good result Pregnancy followed Born at Term
82	Normal	Lacerated	Premenstrual Hemorrhagic	Premenstrual	Lacerated	"	"	"	2 Mths	56	1	D&C Amputation of Cervix Perineorrhaphy Hemorrhoids	Good result No bleeding
85	Retroversion	"	Not stated	Not stated	Moderately Lacerated	"	"	"	1½ Yrs	22	1	Shortening Round Ligaments Appendectomy	Good result No bleeding
88	"	"	Interval	Interval	Not stated	"	"	"	5 yrs	20	1	D&C Shortening Rd Ligts Appendectomy	No report
89	"	"	Not stated	Not stated	"	"	"	Cystic	?	28	2	Shortening Round Ligaments Left oophorectomy	No report
91	"	Normal	"	"	"	"	"	"	?	25	None	D&C Shortening Round Ligaments	Good result No bleeding
94	"	Moderate Laceration	Interval	Interval	"	"	"	"	6 Mths	40	3	D&C Shortening Rd Ligts Appendectomy	Partial success Bleeding less

Summary of Causes of Uterine Bleeding in Cases of Displacement and Laceration – 2 Causes

	CASES	
1	9	Vascular Engorgement in Retroversion 26-55-57-74-85-88-89-91-94
2	4	Focal Infection in Lacerations 37-59-71-82

Displacements.—There are 13 cases of uterine bleeding tabulated in Chart V, where the diagnosis was made of displacement of the uterus and lacerations. The cases of uterine bleeding in this group are the most difficult of interpretation as to the cause of the bleeding. Nine cases had retroversion of the uterus and in four the uterus was normally placed. One of these four had an operation for retroversion four months previously. All of the cases had lacerations either of the cervix, anterior vaginal wall or perineum. The condition of the endometrium and uterine glands was not stated in eight cases. In the five cases subjected to pathologic examination, the endometrium and uterine glands were found of the interval type in three cases, hyperplasia in one case and premenstrual in one case. It is very apparent that there was no pathology found in the endometrium and corpus

glands in these cases. In fact the only pathology found in this group of cases is the displacement of the uterus and laceration, and on this pathology must depend the uterine bleeding.

A few cases may be of interest in detail. Case 26, the first on the chart, was a woman of 28 who had two pregnancies. This patient suffered with uterine bleeding for two months prior to the operation. She has a retroverted uterus and laceration of the cervix and perineum. She had a plastic operation and an external Alexander for shortening the round ligaments. No other pathology was found to account for the uterine bleeding of two months' standing. Case 59 with laceration of cervix and perineum was a woman of thirty years old with uterine bleeding of 6 weeks' duration. No other pathology to account for the uterine bleeding. She was curetted and nothing found in the uterine cavity. A plastic operation relieved her symptoms. Case 94,—retroversion and moderate laceration of cervix. Patient 40 years old, with uterine bleeding of 6 months' duration. A curettage showed an interval mucosa,—no other abnormality, adnexae normal. Shortening of the round ligaments brought relief of symptoms.

What is the cause of the uterine bleeding in this group of cases? Does a retroverted uterus cause uterine bleeding? How are we to explain uterine bleeding in cases of lacerations? We must explain uterine bleeding in cases of retroversion on the basis of vascular engorgement. Emmet called attention to this chronic vascular engorgement in displacements many years ago. The blood supply of the pelvic organs is peculiar to itself, and the anatomic arrangements are such that displacement of the uterus disturbs the physiologic balance between the arterial influx of the blood on one hand and the venous outflow on the other. There results not only a venous engorgement in the uterus but the arterial influx is also disturbed. This vascular disturbance must bring about changes in the endometrium which causes uterine bleeding, though the changes in the endometrium cannot be demonstrated with the microscope. In the cases of lacerations another factor is introduced which must have an important bearing on uterine bleeding, namely, focal infection. The cervix with its many glands as a result of the laceration and influenced by the displacement becomes a prey to the invasion of microorganisms and a chronic endocervicitis results. This focal infection brings about changes in the endometrium, which though undemonstrable, causes the persistent uterine bleeding. On no other basis can the uterine bleeding be explained in this group of cases. In Case 57 there was a uterine bleeding of three months' duration, in a woman 27 years old. The cervix was amputated and round-cell infiltration found. There were no other abnormal findings.

From the study of these cases we must admit that uterine displacement and lacerations are etiologic factors in uterine bleeding, and that the uterine bleeding is due to changes in the endometrium which cannot be demonstrated by the pathologist, due to chronic vascular engorgement and chronic focal infection.

I come now to a class of cases of uterine bleeding, where the cause of the bleeding was put down as ovarian hyperfunction. I shall speak of these cases with a certain amount of diffidence. There were two cases belonging to this class under neoplasm and nine cases in a group by themselves. What is meant by ovarian hyperfunction causing uterine bleeding? The conception is, that as a result of an endocrine disturbance, the ovary functionates beyond a physiologic degree. This hyperfunction of the ovary or corpus luteum keeps the endometrium and uterine glands constantly in a state of hyperplasia and engorgement. In other words the patient keeps on menstruating without a physiologic limit; a physiologic process at first becomes a pathologic one later. In this manner a hyperovarian function becomes an etiologic factor in uterine bleeding.

The history of the two case of hyperovarian function under neoplasm is as follows: Case 1 was a patient thirty-eight years old, with no pregnancies, who was suffering with uterine bleeding for six months. She was operated and a supravaginal hysterectomy and a right salpingo-oophorectomy were done. The pathologist found the endometrium and corpus glands normal except for a hyperplasia. There was a hemorrhagic corpus luteum cyst in the right ovary, the conception being that the diseased ovary was overfunctioning. The second case is identical with the first. The patient was thirty-two years old, no pregnancies, with a metrorrhagia of three weeks' standing. A myomectomy was done and a corpus luteum cyst removed. No other abnormality was found to account for the metrorrhagia. In both of these cases the uterine bleeding was associated with the corpus luteum cyst of the ovary and a possible hyperovarian function.

As said before, I speak of ovarian hyperfunction as a cause of uterine bleeding, with hesitation. An earnest discussion is invited on this subject. Is this a pathologic entity or not? Is it an actual fact that the ovary functionates beyond its physiologic limit or does it only exist in the endocrinologist's imagination? There are very few facts at hand bearing on the ovarian function, upon which argument *pro* and *con* can be based. We do not know in what manner the ovary is influenced by the other ductless glands of the body or *vice versa*. This however we do know, that the product of the pituitary glands affects the uterine tissues most profoundly and we also know that in hypothyroidism with metrorrhagia, the uterine bleeding is promptly controlled by thyroid medication. With these facts before us, it must be admitted that there exists a physiologic endocrine balance, between the ductless glands of the body and a disturbance in this balance may result in an overproduction of hormones in the corpus luteum of the ovary which may keep the endometrium in a state of hyperplasia, resulting in uterine bleeding. Further than this one cannot go into the matter except to state that the therapeutic administration of corpus luteum, ovarian residue, and whole ovary, is at times followed by therapeutic results that are very definite in action.

There are nine cases tabulated in Chart VI where the uterine bleeding was believed to be caused by an overfunction of the ovaries. It will be found by examining Chart VI that in only one, No. 13, of these nine cases was the abdomen opened. In this case a corpus luteum cyst was found enlarged. All the other cases had apparently normal ovaries. However, the ovaries may not be enlarged sufficiently to become palpable in examination and yet manifest a disturbed function. The disturbed function may not rest on a definitely demonstrable change in

Uterine Bleeding
CHART No. VI.
ENDOCRINE DISTRESS 9 CASES
Pathologic Report

No	Endometrium	Uterine Glands	Uterine Tubes	Ovaries	Duration of Bleeding	Age	Number of Pregnancies	Operation	Result
2	Hyperplasia	Hyperplasia	Normal	Normal	6 Months	36	None	Nitric Acid to Endometrium	No Bleeding
13	Premenstrual Myometrium Normal	Premenstrual	"	Corpus Luteum Cyst	" "	30	"	Supravaginal hyst. Left salpingo-oophorectomy	
28	Normal	Indefinite Type	"	Enlarged?	3 Wks	?	Single	D & C	No further Report
38	Hyperplastic	Cystic Glands	"	Normal	5 Months	41	2	D & C Rad. 50 mg 12 hr.	Could not be found
52	Premenstrual	Premenstrual	"	"	4 Months Assn? Menopause	49	None	D & C Rad. 100 mg 24 hr	Private Patient?
60	Hyperplastic	Hyperplastic	"	"	3 Months	48	2	D & C Dilatation & Curettage	No Report
63	Interval Type	Interval Type	"	"	" "	36	2	D & C Dilatation & Curettage	No Bleeding Very good result
64	"	"	"	"	" "	38	3	D & C Dilatation & Curettage	No Bleeding Good result
87	"	"	"	"	9 "	46	Single	D & C	No Report

Summary of Causes of Uterine Bleeding
Hyper-ovarian Function

CONSTITUTIONAL CAUSES
BLOOD DISCRASIE
2 CASES

No			Uterine Bleeding	Operation	Result
16	Chronic Endocarditis	Acute Thyroiditis	2½ Years	Radium 100 mg 24 hrs	Tardy Relief
69	Fibrosis Uteri	Fibrosis of Myometrium	2 Years	Hysterectomy	

Records not complete 2 Cases No 11-34

the ovary itself, but may depend on a disturbance in the physiologic balance between the various ductless glands of the body. In none of the nine cases here tabulated, could one discover any of the other causes of uterine bleeding enumerated before.

Therefore for the want of a better term, and for the want of a better known cause for the bleeding, the cause of uterine bleeding in these nine cases is described as ovarian hyperfunction.

There were two cases of uterine bleeding that were classed under constitutional causes. One was a patient 52 years old with a severe metrorrhagia. She was suffering with an acute endocarditis and an

acute state of hyperthyroidism. There were no local findings to account for the uterine bleeding. Radium brought a tardy relief. The second case tabulated here was one of fibrosis uteri. I have placed this case in this class because the greatest pathologic change is found in the blood vessels. In fact the endometrium was fairly normal. The adnexa were normal. Fibrosis uteri may have an infection as a basis of its etiology, but I could not place the case under infection because it does not conform with the other cases so classified.

The study of the 100 cases of uterine bleeding here reported brings

UTERINE BLEEDING
CHART VII
Causes of Uterine Bleeding in 100 Cases.
14 Causes

1 Abortion, all types.
2 Ectopic Gestation.
3 Low Implanted Placenta.
4 Chorio-Epithelioma.
5 Inflammatory Reaction in the Endometrium
6 Polypoid Condition of the Endometrium.
7 Carcinoma.
8 Sub-Mucous Fibroids.
9 Penetrating Uterine Glands.
10 Degeneration of Uterine Fibroids.
11 Hyper-ovarian Function.
12 Vascular Engorgement.
13 Focal Infection.
14 Constitutional Causes.

out the fact that there were no less than 14 different causes for the bleeding enumerated as shown in Chart VII.

SUMMARY

1. From the study of the pathologic findings in the 100 cases of uterine bleeding here reported, it is found that the cases may be divided into six classes according to the etiologic factors that are causing the loss of blood from the uterus.

2. That the pathologic changes in the endometrium play the greatest rôle in uterine bleeding and that this occurs in 70 per cent of cases divided as follows: In 34 cases infection plays the important rôle in causing endometrial pathology. In 25 cases neoplasm causes the dis-

turbance in the endometrium and in the remaining 11 cases the endometrium is disturbed by a hyperfunction of the ovaries.

3. That the uterine bleeding is caused also by vascular engorgement of uterus and adnexa and focal infection of the generative organs.

4. That pregnancy including ectopic gestation plays a very important rôle in causing uterine bleeding, having been found present in 13 cases.

5. That constitutional causes play a very minor rôle in causing uterine bleeding.

6. That after incomplete abortion the endometrium undergoes the physiologic change incident to menstruation, although the uterine cavity

Uterine Bleeding
CHART VIII

Conclusions

Uterine bleeding
Classified according to Etiology

1st Pregnancy
2nd Infection
3rd Neoplasms
4th Displacement & Lacerations
5th Endocrine Distress
6th Constitutional Causes

Neoplasms 38 Cases
Causes of Bleeding (Seven)

1st Carcinoma	8 Cases
2nd Adenoma polyposum	7 "
3rd Endometritis	11 "
4th Sub-mucous Fibroids	6 "
5th Penetrating Uterine Glands	3
6th Hyperfunction of Ovaries	2 "
7th Degeneration of Neoplasms	1 "
	Total 38

Infection 23 Cases
Causes of Bleeding (Four)

1st Endometritis	11 Cases
2nd Polypoid Condition of Endometritis	4
3rd Various Phases of Menstruation	1
4th Oedema of Endometrium	2
Not stated	5
	Total 23

Uterine bleeding in 70% due to Endometrial Pathology

Infection	34 Cases
Neoplasms	25 "
Hyper-ovarian Function	11 "
	Total 70

Pregnancy 13 Cases
Causes of Bleeding (Four)

1st Abortion	8 Cases
2nd Ectopic	3 "
3rd Malposition of Placenta	1 "
4th Chorio-Epithelioma	1 "
	Total 13

Displacements & Lacerations 13 Cases
Causes of Bleeding (Two)

1st Vascular Engorgement	9 Cases
2nd Focal Infection	4 "
	Total 13

Endocrine Distress 9 Cases
Causes of Bleeding (One)
Hyper-ovarian Function

Constitutional Causes 2 Cases
Causes of Bleeding (Two)

Endocarditis & Thyroiditis	1 Case
Fibrosis Uteri	1 "

may harbor retained secundines which cause continued uterine bleeding as shown in Cases 7 and 36.

7. That in cases of uterine bleeding with history of infection, the bleeding is due to a diseased endometrium, the adnexae remaining perfectly normal. This fact is shown by Cases 3, 30, and 39.

8. That curettage of the uterus is a very important procedure in cases of uterine bleeding, as the bleeding may depend entirely on the condition of the endometrium, in spite of the fact that other pathologic entities may be present, such as diseased adnexa, fibroids, etc. This point was strikingly illustrated in Case 61; the removal of diseased tubes failed to benefit the bleeding, while a curettage brought relief.

9. That in cases of uterine bleeding two or more causes may be operative in the same case at the same time as in Case 29, where there was a

cervical polyp present, a fibroid of the uterus and an incomplete abortion.

10. That adenoma polyposum of the endometrium is responsible for a large number of cases of uterine bleeding, at least 20 per cent, and is a distinct pathologic entity.

11. That when uterine glands penetrate the musculature they may be the cause of uterine bleeding, shown by Cases 17, 20, and 73.

12. That there are a certain number of cases—11 in the 100 cases here reported—of uterine bleeding, where the cause of the bleeding is obscure, where no pathologic changes are found to account for the bleeding, and where we have to fall back upon the theory of endocrin distress. These have been called cases of ovarian hyperfunction.

13. That there are a certain number of cases where the uterine bleeding depends on displacements and lacerations, the bleeding being due to vascular engorgement and "focal infection."

14. That submucous fibroids cause uterine bleeding by bringing about changes in the endometrium overlying the neoplasm.

15. That in a very small number of cases, uterine bleeding may be caused by a degeneration of the neoplasm; the necrosis bringing about cell destruction and escape of blood directly into the uterine cavity.

40 East Forty-first Street.

HEMORRHAGIC LESIONS OF THE PLACENTA AND THEIR RELATION TO WHITE INFARCT FORMATION*

By F. P. McNalley, B.S., M.D., and Wm. J. Dieckmann, B.S.,
St. Louis, Mo.

From the Department of Obstetrics, Washington University School of Medicine.

ONLY a small part of the extensive literature on placental infarcts is devoted to the so-called red infarcts, and this term has been loosely used by different authors to describe different lesions. So far as we can determine there is no generally accepted definition of this term. We have chosen to call all red lesions hemorrhagic and have attempted to distinguish between red infarcts and other conditions which should not be so called, and to demonstrate what relation, if any, they have to white infarcts.

Our routine method of examination of placentae is as follows: They are washed free of blood and placed in 10 per cent formalin and allowed to remain in this solution about seven to ten days. They are then described in gross and cut in slices about 2 cm. thick. This enables us to detect lesions in the substance of the organ that would otherwise be missed. Blocks are taken for microscopic study from all areas showing anything unusual.

Up to the time this work was started we had examined three hundred and twenty placentae, at or near full term, and were struck with the frequency of hemorrhagic lesions, which were noted in one hundred and twenty-three cases, or 38 per cent. These have been variously termed: apoplexy, (Cruveilhier, Jacquemier, Gierse, Meckel, Scanzoni and Williams); hematoma, (Klebs); hemorrhage, (Kuehnel, Simpson, Rokitansky, Brindeau and Nattan-Larrier); hepatization, (Bracket and Scanzoni); red infarcts, (Williams, Young and others).

The terms "apoplexy" and "hematoma" seem to have been given to dark-red, circumscribed areas. Williams states that he has had occasion to examine them in but a few instances, but believes they differ essentially from other forms of infarcts. Kuehnel seems to have the same lesion in mind when he describes hemorrhages into preformed cavities. Brindeau and Nattan-Larrier give three forms of hemorrhage: The first, a hemorrhagic nodule formed by hemorrhages of varying size into the substance of the placenta, the apoplexy of other authors; second, diffuse hemorrhage in the substance of the placenta, apparently the hepatization described by others; third, retroplacental hemorrhage.

*Read before the St. Louis Gynecological Society, April 14, 1922.

The term "red infarct" is applied by Williams to two different lesions. First, pinkish or brick-dust colored masses, irregularly shaped, more or less solid, and sharply marked off from the surrounding tissue and occasionally occupying a large portion of the placenta; and second, round areas varying from bright red to almost black in

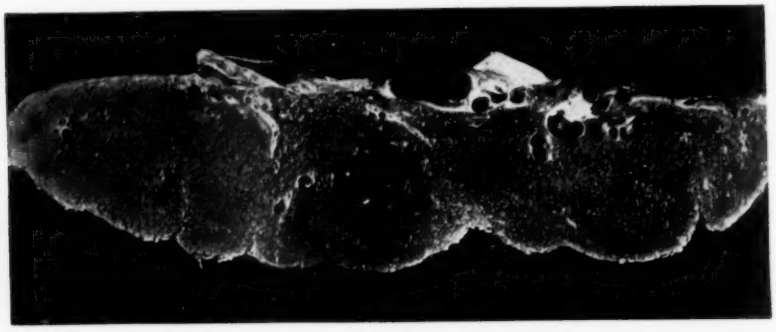

Fig. 1.—Showing two types of hematomata, the large circumscribed one in the center and at either end the smaller ones not circumscribed, as described in text.

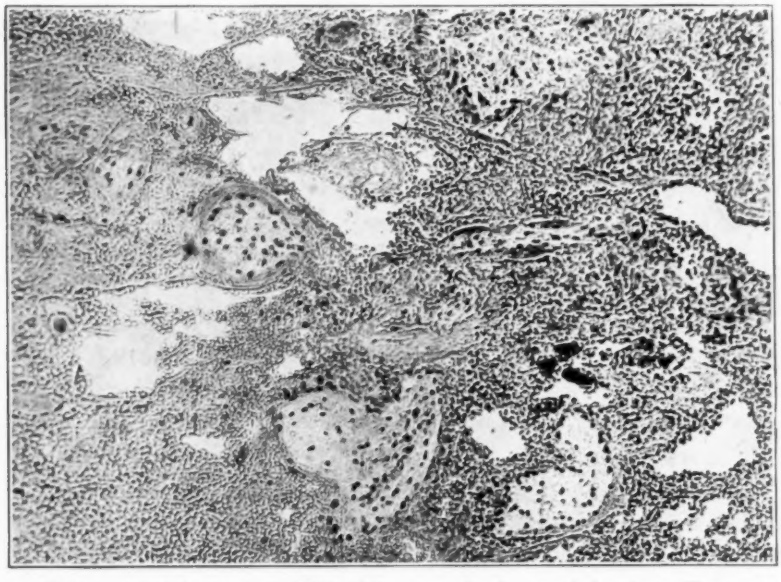

Fig. 2.—Thirteen weeks' abortion. Villi without vessels showing infarction. They are widely separated by marked hemorrhage.

color, which are apparently composed almost entirely of blood and are sharply marked off from the surrounding tissue by a capsule which presents a more or less fibrous appearance. These have also been spoken of as apoplexy. The first kind, he says, is closely related to the ordinary white infarcts, and differs from them only in the fact that a greater number of red blood cells are enclosed in the meshes of the fibrin network by which the degenerated villi are bound to-

gether. We have not found anything in our study to correspond to this description.

The red infarcts of Young are entirely different from those of Williams. He believes that white infarcts are red in the beginning. He says, "In the earliest stage recognizable to the naked eye the patch is deep red, purple, or even black. It may not be visible until thrown in relief against the surrounding paler placenta which has lain for some days in a fixing solution into which the unclotted blood of the intervillous spaces has oozed. Microscopically in this stage the color is due to the engorgement of villi with blood. The whole appearance in this stage closely simulates the vascular hyperemia and stagna-

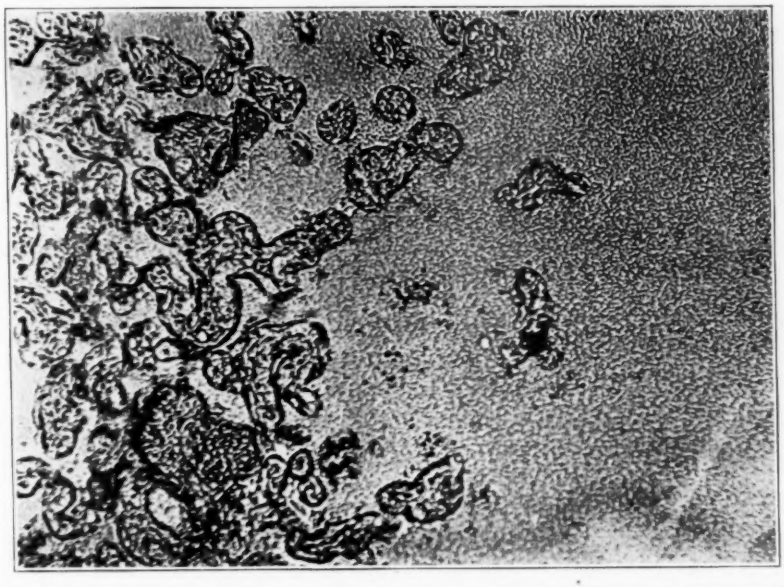

Fig. 3.—Microscopic picture of the smaller hematomata shown in Fig. 1, showing practically normal villi surrounding blood.

tion which occurs in other tissues when there is any lowering of vitality. It is an evidence of a local reaction which precedes the local death that is inevitable when the blood supply is cut off. As the process goes on the infarct becomes paler and finally white, due to the disappearance of the hemoglobin. In association with these vessel changes there are other changes in the infarct. The villi are closely packed together, the maternal spaces being empty or nearly so, the villi lie opposed to one another, or there may be actual fusion of the epithelial surfaces. Eden explained this as being due to a progressive diminution of blood supply to a part of a cotyledon by obstruction of a maternal artery, which would cause the villi to become crowded together because there would not be sufficient blood to maintain their normal distance from one another."

That obliteration of maternal arteries does take place before labor is shown by Goodall in his work on involution of the uterus. He describes changes in the arteries of the uterus of a woman forty-four hours after labor that must have taken days for their development. "The changes present in the vessel walls, particularly in the elastic coats during involution, are present, but have gone on to a complete

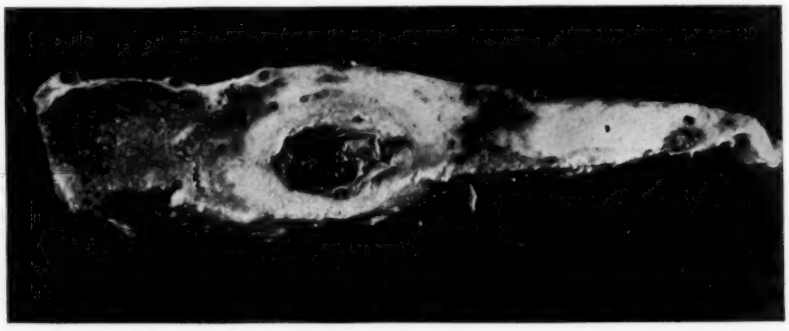

Fig. 4.—Large hematoma surrounded by white infarct, most of which is formed from villi.

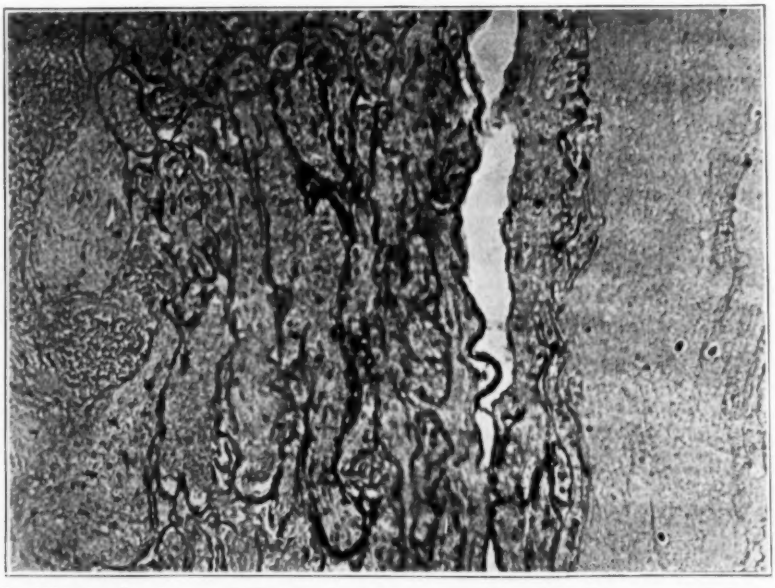

Fig. 5.—Microscopic picture of Fig. 4, showing, to one side, white infarct formed from villi, and to the other, fibrin formation in blood clot, resembling white infarct.

obliteration of the lumen or a narrowing of its calibre by these changes and by thrombus formation." He goes on to say, "That there is a close connection between these changes and the presence of so-called placental infarcts, I have but very little doubt. The presence in such a specimen as this, in numbers much greater than in the average parturient uterus, is quite in accord with the multiplicity of

placental infarcts found in placentae of women suffering from renal disease." If obliteration of an artery can affect an isolated portion of the placenta, it means that different portions of the placenta are nourished by different vessels and there is not a free communication of the maternal sinuses. Young believes this is so because injection of coloring matter in the center of a lobule will demonstrate its isolation and also because the fissures between the cotyledons are occupied by solid partitions of decidua.

Young believes that the villi are not dependent on their own vessels for nourishment but on the maternal blood, and gives as his reasons: (1) The time when the chorionic elements are most active and proliferate most rapidly is during the early stages of development and before there are any fetal vessels formed. (2) In hydatid mole the villi proliferate and there are no fetal vessels. (3) In tubal pregnancy where blood supply from the ovum has been cut off by hemorrhage, but where the maternal circulation is unimpaired the tips of the necrotic villi remain healthy. He states that retroplacental or intraplacental hemorrhages are the cause of infarct formation because they do interfere with the maternal supply to the affected part, but does not apply the term "red infarct" to them.

It is not our purpose in this paper to take up the whole subject of white infarct formation and we include this work of Young because in our own study we found one "hemorrhagic lesion" that answers Young's gross description and three others that answer his microscopic description. One small cotyledon, 3 cm. in diameter, at the margin of the placenta was dark red in color and rather firm, with a slightly paler area about ½ cm. wide at one margin. Microscopically the dark red portion showed the engorgement of the villi and close packing of the villi described by Young, and the paler portion showed his later stage, the hemoglobin less in amount, cells taking the stain poorly and the villi in places fused and in places showing a thin layer of fibrin about their periphery, such as can be seen in almost any infarct.

Besides the lesion above described, we have found three other types. (1) Cavities, varying in size from that of a pinhead to ½ cm. These are always irregular in outline, with no clear-cut margin, and have the appearance of being formed by wide separation of the villi. Most of these contain blood but a few are empty. Of the hundred and twenty-three placentae showing hemorrhagic lesions, we found eighty-seven with these cavities, or 70.7 per cent (Fig. 1.) Microscopically they bear out the appearance described in gross, i.e., there is no clear-cut margin, the cavity having the appearance of being formed by a wide separation of the villi, with the latter protruding into it much like the branches of a tree. The blood, when present,

has the appearance of fresh blood, there being no tendency for fibrin formation such as is seen strikingly in our third type of lesion. Except in one case, infarct formation in the surrounding villi is practically absent. In this case the presence of normal vessels in the villi is very noticeable, showing that infarct formation is possible without there being an endarteritis. This has been very noticeable in all the white infarct formation involving villi and associated with hemorrhagic lesions.

We have also found infarcted villi in a very early placenta associated with hemorrhage. This was from a woman thirteen weeks pregnant who took a rough automobile ride three days before abortion. The specimen in the gross consisted of a mass of placental tissue

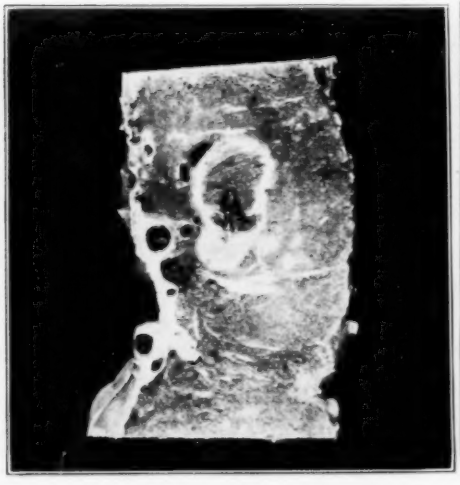

Fig. 6.—Early stage of transformation by fibrin formation of a blood clot, as seen in Fig. 1, into what resembles white infarct in gross.

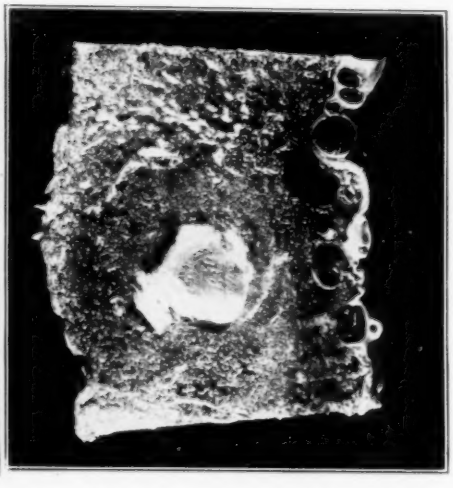

Fig. 7.—Later stage of same process seen in Fig. 6.

8 cm. × 2 cm. On section it was densely infiltrated with blood, making it impossible to recognize placental tissue. Microscopically it showed small numbers of villi which showed infarction in all stages. This is a case (Fig. 2) showing infarction where there are no fetal vessels, the maternal supply cut off by hemorrhage, probably traumatic.

Our second type is that in which the whole, or a large part of the placenta is densely infiltrated with blood, the organ being heavy and very thick in comparison to other placentae; it is spongy and oozes dark red, sometimes almost black blood, and to this the term "hepatization" might well be applied. This condition we found in eight cases, or 0.65 per cent of the hundred and twenty-three. In seven of these cases we also found the cavities first described. Microscopically

they show nothing characteristic except that all the intervillous spaces are large and filled with blood.

The third type, and the one which we believe is probably the same as the first but more extensive and which plays an important part in

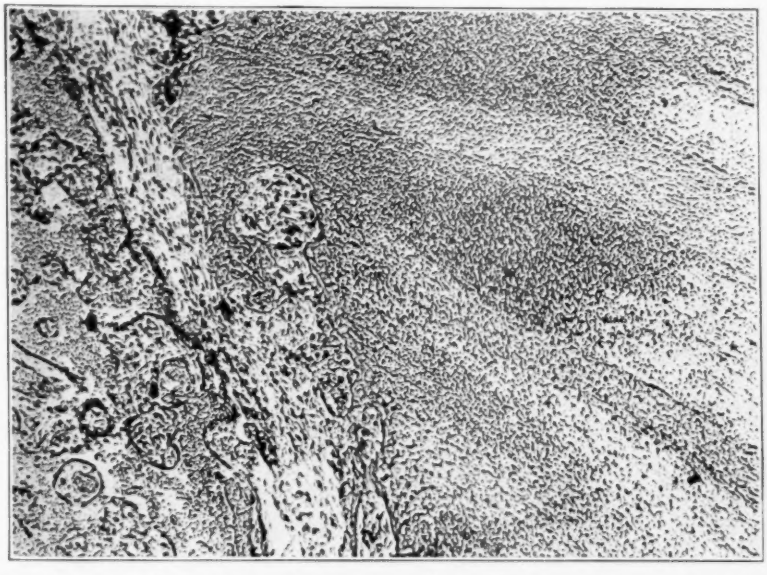

Fig. 8.—Microscopic picture of Fig. 7. Layers of fibrin with spaces between filled with red blood cells.

Fig. 9.—A very large hematoma being converted into white infarct. Notice white striations of fibrin being laid down in the blood clot.

the formation of at least some white infarcts, occurred in forty-three cases, or 35 per cent. These correspond to one kind of Williams' red infarcts, the apoplexy of other writers, and the intraplacental hemorrhages of Young. We propose to show that starting

with a collection of pure blood these become converted into structures that in the gross have the identical appearance of white infarcts, but in whose formation villi play no part.

These collections of blood differ materially from those first described in that they are always larger, varying from 0.5 to 3 cm. in diameter, some irregular in shape, but many round or oval. They are definitely circumscribed, having a clear-cut margin and early show nothing like a capsule, in the gross appearing merely as a collection of blood surrounded by normal placental tissue. In this stage microscopically they show merely a blood clot, the surrounding villi being normal or showing slight white infarct formation. This infarct formation may remain slight or there may be a large area of surrounding villi that undergoes the changes described by Young and forms a white infarct. This white infarct formation seems to be explained by Young's theory of interference with the maternal supply by the collection of blood. Where it is not so extensive, there al-

Fig. 10.—Hematoma just beginning to show fibrin formation.

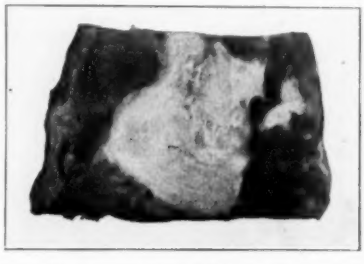

Fig. 11.—A mass identical in appearance with white infarct, formed from a hematoma, which, in gross cannot be distinguished from the ordinary white infarct. This is from the same placenta as Fig. 10.

ways being some, we assume the adjacent areas to be supplied by other vessels. We do not believe this appearance of blood surrounded by infarcted tissue can be called a hemorrhage into white infarct as has been claimed by some authors.

Independent of this infarction the collection of blood itself undergoes the changes which convert it into a mass analogous to white infarct in the gross. This is in accord with the findings of Dieulafoy who says, "The lesion is characterized by the appearances of hemorrhages, which are converted into white infarct. The primary lesion is hemorrhagic; the white infarct is secondary. The recent infarct contains fluid blood, or having a gelatinous consistency, is almost black in color. When the infarct is older it becomes pale, the coloring matter of the blood disappears and finally reaches the tint of a white infarct. If the infarct is incised it appears to be formed of fibrin,

more or less dense in appearance and sometimes disposed in a series of concentric layers. In the center we often find a small collection of colored fluid forming the residue of the blood serum."

Cruveilhier in his article on apoplexy stated that he had observed a complete series of cases which demonstrated the transformation of ordinary apoplexy into the pale, white, fibrous areas. Shaffer considers that many of the small subchorial infarcts are the result of the organization of localized hemorrhages. De Lee says, "organization of the clot occurs and it is possible to trace the changes through the stages of (1) soft, red-black clot; (2) brick-red, firmer masses; (3) fibrin formation in the periphery; (4) through and through hard, fibrous nodule."

Fig. 12.—Microscopic picture of Fig. 11, showing fibrin with few red blood cells caught in its meshes. No evidence of any villi.

Williams disagrees with these authors and thinks these lesions differ essentially from other forms of infarcts, believing endarteritis is the chief etiological factor in white infarct formation.

In the fresh collection of blood already described there appear striations near one margin, slightly lighter in color than the remainder of the clot. These increase in number and become paler, giving the whole clot a pink rather than a red color, with white striations throughout. As the process goes on the color becomes whiter, the striations become wider and closer together until, as in one of our specimens, it has the identical appearance in gross of a white infarct (Fig. 11). The microscopic appearance corresponds with the gross. The striations are layers of fibrin being formed, the red blood

cells are becoming fewer and the fibrin more abundant, giving the appearance of a laminated clot. We are able to follow the decrease in red cells and the increase in fibrin throughout the progressive stages to the typical white infarct in gross which still shows a few red cells caught in the infarct. There are no villi involved in this process, the blood itself being converted into the white infarct. The different stages, both gross and microscopic, are shown in Figs. 3 to 12. There is no evidence in any of our sections of organization as it is usually understood, i.e., proliferation of fibrous tissue and ingrowths of new vessels.

That hemorrhage can be the origin of white infarct we believe is even more clearly shown in another specimen, a white infarct, 3 cm. in diameter, on the maternal surface which, on section, is seen to

Fig. 13.—This was originally a retroplacental hemorrhage which on the surface looks like the usual white infarct. Notice small clot still remaining in center.

extend 1 cm. beneath the surface. In the center of this infarct is a blood clot 1 × 0.5 cm., seen in Fig. 13. Microscopically (Fig. 14) this clot is seen surrounded by layers of fibrin in which a few red blood cells can still be distinguished. Separating this infarct from the placental tissue about its entire margin is a layer of decidual tissue, the picture one sees in a retroplacental hemorrhage, which condition we believe this infarct originally was, being converted into a white infarct by the process described above, leaving the small collection of blood still in its center.

We believe that the three types of lesions above described have a common etiology.

In considering the question as to the origin of these collections of blood, the first thing to be determined would be whether it is fetal or maternal in origin. If fetal, it is evidently due to rupture of a fetal

vessel and is a true hemorrhage. Brindeau and Nattan-Larrier have observed this but we have not been able to confirm it. If, on the other hand, it is maternal blood, such a term could not be applied, for, as Williams says, "The intervillous spaces are in themselves blood vessels, and we are hardly justified in speaking of hemorrhage into blood vessels."

We have not been able to definitely decide the origin of the blood but feel at the present time it is probably maternal. If such is the case, the most logical explanation would be back pressure due to venous stasis from thrombosis, the size of the lesion being in proportion to the extent of inference with the return flow. This is pointed

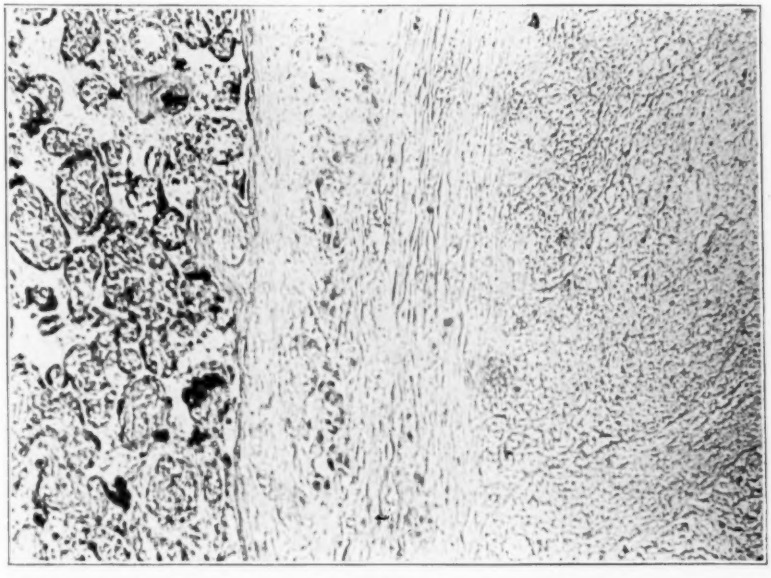

Fig. 14.—Microscopic picture of Fig. 13, showing villi on one side, fibrin on the other, and between decidual cells.

out clearly by Young and advanced by him as the origin of retroplacental hemorrhages. Goodall, in speaking of changes in the veins before labor, says, "In the smaller veins of the placental area there is usually a complete obliteration due to the building of a thrombus." Schilling believes that in some cases there is a thrombosis originating in the maternal vessels of the decidua and then gradually spreads to the intervillous spaces.

In all our cases there were only four of toxemia, one of which is mentioned above. Aside from this one the lesions were no different from those found in our normal pregnancies. It is interesting to note that many authors find "red infarcts" and albuminuria closely associated. Rouhaud, in 1882, noted that 40 per cent of the cases which had albuminuria had red infarcts and 40 per cent of these had dead

infants. Many other authors have shown similar relationship. There were four stillborn infants in our series, two of which were in toxemic cases and the other two accounted for by other factors. These cases had lesions similar to the others where the infants were in good condition.

CONCLUSIONS

(1) At least some of the usual white infarcts resulting from changes in the villi may be red in color at first as described by Young. To this the term "red infarct" is applicable.

(2) The other lesions that have been variously termed should not be called red infarcts, because in reality they are not infarcts but collections of blood, to which the term "hematoma" or "hepatization" might be applied, depending on whether circumscribed or diffuse. They have a common etiology.

(3) The maternal blood is of primary importance in nourishing the villi, as pointed out by Young.

(4) Collections of blood in the placenta may be the beginning of white infarcts in two ways, (a) by the actual conversion of the blood itself into a white infarct, which in the gross does not differ from any other; (b) by causing infarction of the surrounding villi as the result of interfering with maternal circulation.

(5) There can be white infarct formation without endarteritis in the fetal vessels. Whether this is true in all infarcts we are not prepared to say at the present time.

REFERENCES

Brachet: Quoted by Williams. *Brindeau and Nattan-Larrier:* Quoted by Williams. *Cruvielhier:* Quoted by Williams. *De Lee:* Textbook of Obstetrics. *Dieulafoy:* Textbook of Medicine, Translated by Collins and Lieberman, ii, 1904. *Eden:* Jour. Path. and Bacteriol., Edinburgh and London, 1897, iv, 265. *Gierse:* Quoted by Williams. *Goodall:* Studies from the Royal Victoria Hospital, Montreal, ii, No. 3. *Holland:* Jour. Obst. and Gynec. of the British Empire, 1909, xvi, 255, 325, 384. *Jacquemier:* Quoted by Williams. *Klebs:* Quoted by Williams. *Meckel:* Quoted by Williams. *Rokitansky:* Quoted by Williams. *Scanzoni:* Lehrbuch der Geburtshülfe, 1853, ed. 2. *Simpson:* Edinburgh Medical Journal, vol. xlv, p. 266. *Shaffer:* Quoted by Williams. *Talbot:* Surg., Gynec. and Obst., 1921, xxxii, 552. *Young:* Jour. Obst. and Gynec. of the British Empire, 1914, xxvi, 1.

503 WALL BUILDING. (*For discussion see p.* 97.)

BLOOD STUDIES IN NORMAL PREGNANCY*

By O. S. Krebs, B.S., M.D., and A. P. Briggs, B.S., St. Louis, Mo.

(From the Department of Obstetrics, Washington University School of Medicine.)

THERE has been, probably, no more fertile field for investigation than that offered by various abnormal conditions that present themselves during pregnancy. Just one of them, eclampsia, has a very extensive literature dealing with the many theories of its causation, but as yet the exact nature of the underlying cause is not known.

Various investigators tell us of the value of urine study in the toxemias of pregnancy; others have emphasized the rather questionable value of a determination of the nonprotein nitrogen in contrast to the clinical course, blood pressure and urinary findings; another has pointed out the value of the noncoagulative nitrogen coefficient; and still others mention that high nonprotein nitrogen of which the urea nitrogen forms a small percentage, is characteristic of toxemia of the hepatic type; and finally others say that the blood pictures in eclampsia and toxemia are interpreted most readily in terms of kidney insufficiency. Thus we find that blood studies which have been confined largely to the nitrogenous constituents are valuable in considering the diagnosis and prognosis of the various disorders of pregnancy but throw no light on the exact nature of the toxemia, or the source of the toxins.

Another line of investigation seemed reasonable to assume as perhaps being able to throw some light on the various abnormal conditions that obtain during pregnancy. An investigation of the inorganic constituents of the blood was therefore proposed, the investigation being reported having taken into consideration bloods of the normally pregnant woman.

A review of the literature, as can be seen from the following, has shown that the inorganic constituents of the blood in pregnancy have been rather indifferently investigated. Calcium and magnesium have been the elements most frequently determined and the nephritides were the favorite clinical sources of the bloods; the toxemias of pregnancy being rather superficially studied.

In 1906, Silvestri estimated that the calcium content of a fetus increases rapidly toward the end of term and feels that this must be a great drain upon the mother, especially during that time.

*Read before the St. Louis Gynecological Society, April 14, 1922.

In 1908, MacCallum and Voegtlin[1] in studying the relation of tetany to parathyroids, say that in osteomalacia and rickets there is evidently a common source which leads to the decalcification of the bones and other tissues and consequently to tetany. Whether this depends on a lesion of the parathyroids as Erdheim suggests, remains to be determined. In the tetany which accompanies pregnancy and lactation, however, it can easily be believed that there is no organic lesion of the parathyroid glands, but that the decalcification of the tissues is directly due to the extraordinary drain upon them in the production of the bones and other calcium rich tissues of the fetus, or in the secretion of milk which has a large calcium content. Further, they say that numerous researches have shown the important relation of the calcium salts to the excitability of the central nervous system.

In 1910, Mitchell[2] reported a new theory of eclampsia on the basis of calcium deficiency. He says that the calcium theory explains many clinical features of eclampsia, viz., the greater frequency at term; the greater frequency in twin pregnancy; the nervous excitability and convulsions; the delayed coagulation time of the blood; the decayed teeth; the urinary findings; the associated edema; the eclampsia of lactation and the newly born; the well-known cessation of convulsions under mild diet.

In 1916, Howland and Marriott[3] investigating the calcium content of the blood in rickets and tetany stated that in the majority of normal cases the calcium varies between 10 and 11 mg. per 100 c.c. of blood serum.

In 1916, Marriott and Howland[10] in studying the acidosis of nephritis in regard to phosphate retention in the blood, said that the retention of acid phosphate would seem to be sufficient to account for the degree of acidosis observed. That the high phosphate content is due to interference with a specific function of the kidneys and not to increased phosphate production in the body or increased absorption from the intestinal tract is shown by the fact that the urinary output of phosphate is not increased and indeed may be diminished. The accumulated phosphates of the serum, even though neutralized, are capable of doing serious harm to the organism. The amount is sufficient to have a definite influence on the osmotic pressure. This suggests a possible connection with the phenomena of edema and hydremia. Marriott and Howland have found further, in most of the cases studied, a marked reduction in the calcium of the serum. What influence this low calcium content may have in the production of such symptoms as convulsions and hemorrhages can only be suggested. The low calcium content they refer to the excess of phosphates in the plasma. They say that it has been shown repeatedly that phosphates administered in any form cause an increased elimination of calcium, chiefly by way of the intestines; the converse is also true.

In 1917, Lyman[4] reported the average calcium content of the blood for males as 6.1 mg. per 100 c.c. of blood, while that of females was 7.1 mg.

In September 1917, Halverson, Mohler and Bergeim[11] published an article on the calcium content of the blood serum in certain pathological conditions. In pernicious vomiting of pregnancy they found the value for serum calcium was normal, the excretion of calcium, ammonia and acid was very high. After alkali administration there was an immediate drop in calcium excretion to less than 10 per cent of the normal value. Similar rapid decrease in acidity and ammonia were noted. Apparently calcium excretion increased with an excessive acid production. Distinct decreases in calcium content were noted in cases of hematogenous jaundice, eclampsia, pneumonia, and particularly uremia. The normal range was from 9 to 11 mg. of calcium per 100 c.c. The authors emphasized the great constancy of calcium in the serum as compared with most blood constituents.

In 1920, Denis and Minot[5] concluded that the result of a study of the effect

of the administration of calcium salts by mouth to men, cats and rabbits indicates that in most cases it is impossible to increase the concentration of calcium in the plasma by injection of calcium salts, but that in cats and rabbits where the initial concentration is low, it is sometimes possible to greatly increase the amount of calcium in the plasma by feeding salts of the element.

In 1920, Underhill, Honeij and Bogert[6] in studying calcium and magnesium metabolism in disease, stated that it is evident that with normal subjects in negative calcium balance, added calcium causes the balance to become positive, and that the additional calcium exerts little or no influence upon the magnesium balance. The influence of added calcium is, however, only temporary, since its withdrawal results in a resumption of a significant negative calcium balance. Added magnesium changes a slightly negative magnesium balance to a strongly positive one without a very significant influence upon the calcium balance.

Blair Bell,[7] in "The Sex Complex," 1921, says concerning calcium metabolism that it is differently employed at three different periods of life. Until puberty, calcium is chiefly utilized in building up the skeleton; after this epoch, for reproduction, and lastly and pathologically, if the termination of life be pathological, for producing changes which are associated with senility, namely, the retention of lime salts in the tissues, especially the arteries. The calcium metabolism is directly influenced by most of the internal secretions. Some, such as infundibulin and suprarenin, induce the retention of calcium salts in the blood and tissues—that is to say, they are anabolic in respect to these salts; while others, such as the thyroid and ovarian secretions, are katabolic. Consequently, as lime salts themselves have a pressor action, an indirect effect of this nature is produced on the vasomotor system by those secretions which lead to the retention of calcium compounds in the blood quite apart from their own specific actions. Considering parathyroid secretion, Bell says that all that concerns the calcium metabolism, closely concerns the reproductive functions; consequently the parathyroids may be involved in many disorders of menstruation, pregnancy, and lactation, although as yet there is little direct evidence that this is so. Erdheim has stated that in osteomalacia he has found hyperplasia of the parathyroids. If this exceed that found normally in pregnancy, it is probable that this change indicates an attempt on the part of these organs to prevent the excessive secretion of lime salts which occurs in osteomalacia, for it has been shown that after removal of the parathyroids there is a deficiency of calcium salts in the tissues. Tetany of pregnancy is attributed to insufficiency of parathyroid secretion and the experiments of Vassale and Generali, and Adler and Thaler, appear to bear out this view. These observers found that partial parathyroidectomy may give rise to no symptoms unless the animal be pregnant, when tetany is prone to occur, or to follow parturition. It is, therefore, more than probable that the tetany which may be seen in the human subject during the later stages of pregnancy is associated directly or indirectly with the metabolism of calcium and the control of this metabolism by the parathyroids.

Vassale believes that insufficiency in the parathyroids is to be found in eclampsia,—a toxemia which he thinks may be successfully treated by an extract of these organs. It seems likely that this method of treatment acts indirectly—that is, calcium retention is promoted and excessive acidosis, so constant in this disease, is prevented. It has been suggested by Silvestri that excessive vomiting of pregnancy may be due to suprarenal insufficiency, and this writer claims to have treated hyperemesis successfully by the administration of suprarenin. If this be so, the therapeutic effect is somewhat complicated; the suprarenal being damaged by the toxemia of pregnancy allows an excessive excretion of lime salts,

and the loss of calcium may induce a condition of acid intoxication with associated vomiting. Again, it will be remembered that Bossi has shown that osteomalacia may be relieved by injections of suprarenin, and that this author concluded that in this disease there is a suprarenal insufficiency.

In 1921, Myers and Short[8] stated that the potassium content of normal human blood serum amounts to rather less than 20 mg. per 100 c.c., while for whole blood the figures are from 8 to 12 times this amount. In a series of seven nephritics with marked nitrogen retention, no increase in potassium content of the serum or whole blood was noted. The authors say that these few observations do not lend support to the suggestion of Smillie that some of the symptoms of uremia may be due to potassium poisoning as a result of retention of this element.

Kramer and Tisdall[9] in the same Journal, reported values for various of the inorganic constituents of the blood. These figures were: Sodium, 175 to 225 mg. per 100 c.c.; potassium, 153 to 201 mg.; calcium, 5.3 to 6.8 mg.; magnesium, 2.3 to 4.0 mg. The concentration in the normal blood varies more than in normal serums due to variation in the corpuscular content of the blood.

The data we have obtained are in the nature of a preliminary report to studies we expect to carry on at some future time. Our cases were women with clinically normal pregnancies, taken at various periods of gestation. Normal cases were studied first so that a comparison could be drawn with the study of pathological bloods which we hope will follow. In conjunction with a determination of the inorganic constituents, certain of the nitrogenous elements and sugar content were estimated, so that if at any time any unexpected interpretation might be suggested that we would have the figures of a rather complete blood study for possible corroboration.

Table I shows the tabulated figures in our series.

The inorganic determinations were made in the Department of Biological Chemistry by A. P. Briggs. Sodium was estimated by the gravimetric method of Kramer[12]; potassium by an unpublished colorimetric method by E. A. Doisy of the Department of Biochemistry, Washington University School of Medicine; calcium by a volumetric permanganate-oxalate method; magnesium by a colorimetric method by A. P. Briggs (now in press); phosphorus by a modification of the colorimetric method of Bell and Doisy[13]; chlorides by a volumetric method of McLean and Van Slyke.[14] The nitrogenous elements were determined by the Folin-Wu[15] methods and sugar by the Shaffer-Hartmann[16] technic.

The results of the study of blood with special reference to the inorganic constituents shows a great constancy of all the elements in normal pregnancy, regardless of the period of gestation, with one exception, namely, that in the last weeks of pregnancy our few cases showed the calcium content to be slightly lowered. This latter finding is in accord with the ideas of MacCallum and Voegtlin, who emphasize the drain on the maternal organism by the demands of the fetus and in lactation. Halverson, Mohler and Bergeim mention

TABLE I

CASE	AGE	GRAVIDA	GESTATION	URINES: ALBUMIN	URINES: SUGAR	BLOOD PRESSURE	N. P. N.	UREA	URIC ACID	CREATININE	SUGAR	SODIUM	POTASSIUM	CALCIUM	MAGNESIUM	PHOSPHORUS	SODIUM CHLORIDE
I	21	I	8	0	0	108/60	23.7	10.3	3.0	1.7	.078%	311	21.3	9.9	2.69	2.16	587
II	29	III	8	0	0	115/75	27.6	10.0	3.1	2.1	.079%	296	....	10.0	2.5	2.93	575
III	25	II	9	0	0	98/54	30.7	11.8	2.0	2.3	.111%	309	19.9	9.3	2.2	3.2	625
IV	31	III	11	0	0	96/68	21.5	9.5	3.3	2.2	.....	...	20.9	9.7	2.5	3.2	576
V	28	III	12	0	0	98/50	24.4	....	2.5	2.7	.107%	289	24.1	9.3	2.35	2.9	575
VI	29	VIII	16	0	0	98/62	30.0	10.3	2.6	2.7	.....	334	24.0	9.05	2.18	2.4	620
VII	29	III	16	0	0	108/62	30.0	10.5	3.1	1.8	.113%	286	28.6	7.5	2.12	1.08	563
VIII	14	I	18	0	0	122/60	31.5	9.7	3.3	1.8	.100%	289	22.9	7.8	2.15	1.39	576
IX	20	III	21	0	0	104/48	29.4	....	3.4	2.6	.123%	305	26.2	9.6	2.3	3.3	600
X	24	III	24	0	0	120/55	34.3	10.5	...	2.0	.085%	327	22.3	9.25	2.64	2.7	595
XI	28	III	26	0	0	128/70	25.0	10.2	3.8	2.5	.122%	...	....	9.35	....	3.8	525
XII	18	I	28	0	0	110/68	24.0	11.1	3.2	2.0	.078%	318	18.3	9.66	2.26	2.4	600
XIII	28	I	34	0	0	88/40	31.5	9.3	2.6	1.9	.124%	323	26.1	8.8	2.2	3.06	617
XIV	16	I	37	0	0	110/55	30.0	9.2	3.3	2.4	.071%	319	24.7	9.36	2.5	2.51	587
XV	15	I	40	0	0	102/62	34.2	....	2.3	2.1	.103%	293	....	8.1	2.44	2.96	567
XVI	30	V	40	0	0	112/64	40.0	11.1	3.5	2.3	.118%	301	23.8	9.0	2.32	3.24	565
XVII	22	I	40	1	0	135/100	26.0	8.6	4.2	2.4	.085%	...	25.7	8.45	2.68	3.3	581

the constancy of calcium and Myers and Short the constancy of potassium in normal bloods.

REFERENCES

(1) Jour. Exper. Med., 1909, xi, 118. (2) Med. Rec., 1910, lxxviii, 906. (3) Am. Jour. Obst., lxxiv, p. 541. (4) Jour. Biol. Chem., 1917, xxx, 1. (5) Jour. Biol. Chem., 1920, xli, 357. (6) Jour. Exper. Med., 1920, xxxii, 41. (7) The Sex Complex, 1921. (8) Jour. Biol. Chem., xlviii, 83. (9) Jour. Biol. Chem., xlviii, 223. (10) Arch. Int. Med., 1916, xviii, 708. (11) Jour. Biol. Chem., 1917, xxxii, 171. (12) Jour. Biol. Chem., 1920, xli, No. 2, p. 263. (13) Jour. Biol. Chem., 1920, xliv, No. 1. (14) Jour. Biol. Chem., 1915, xxi, p. 361. (15) Jour. Biol. Chem., 1919, xxxviii, No. 1. (16) Jour. Biol. Chem., 1921, xlv, No. 2.

(*For discussion see p.* 98.)

RENAL COLIC ASSOCIATED WITH URETHRAL CONDITIONS IN WOMEN*

By E. MacD. Stanton, M.D., F.A.C.S., Schenectady, N. Y.

THE patients comprising the group of cases which I wish to describe have long been recognized by gynecologists and urologists as presenting attacks of typical renal colic but with rather indefinite pathologic findings. Some surgeons, notably Hunner of Baltimore, would have us believe that the pain in many of these cases is due to stricture of the ureter. Hunner believes that true stricture of the ureter is actually a common condition and that our failure to diagnose these cases properly is simply because we fail to recognize the stricture. The attitude of Hunner on this subject was clearly stated in his paper before the New York State Society meeting in 1919, when he said, "In the three and one half years since my first paper, I have diagnosed and treated about 500 cases, and I have had the satisfaction of instructing or inspiring other men in different sections of the country to take up this work and they have obtained similar results. It has been a source of interest to receive letters from some of these men, saying they are finding so many cases that they doubt the accuracy of the technic. I have had the same doubts and misgivings many times in my own work, and it is only because of the satisfactory results to the patient that I am still firm in my belief in the accuracy of my conclusions. I believe I have demonstrated that ureteral stricture is one of the commonest causes of abdominal symptoms in women."

I for one cannot agree with the above statements. In the first place a true stricture is a definite pathologic condition. Real strictures if they exist for any length of time are accompanied by the secondary results of the obstruction, namely, dilatation of the ureter or pelvis

*Read before the Ontario County Medical Society at Clifton Springs, N. Y., April 11, 1922.

above the stricture. Hundreds of thousands of autopsies have been performed with the minutest care by most competent pathologists. Were true ureteral stricture a common condition, it would long ago have been recognized as such through autopsy findings. As a matter of fact, simple strictures of the ureter are not commonly found at autopsy. Hydronephrosis or hydropyonephrosis is rather frequently found postmortem but in the great majority of cases it is due to prostatic obstruction, pressure of tumor growths, stone or other pathology not primarily caused by ureteral stricture.

With modern diagnostic methods, true strictures of the ureter are readily recognized. The findings, obtained by the use of the ureteral catheter and the injection of x-ray opaque fluids into the urinary tract are positive and for actual strictures are definitely characteristic. During the past ten years we have made routine use of the combined x-ray and cystoscopic diagnostic method in practically all cases presenting symptoms referable to the urinary tract. During this time our findings as regards the frequency of gross obstructive lesions of the upper urinary tract agree with what we would have *a priori* expected from previously reported autopsy series. We have encountered a considerable number of cases of hydroureter and hydronephrosis due to such causes as stone, pregnancy, pressure of tumor growths on the ureter and to anomalous vessels in the region of the pelvis of the kidney. Tuberculous strictures have been fairly common. All told, the above-mentioned causes including stone have accounted for less than half of the cases referred to us with undoubted histories of pain of renal origin. Colon bacillus pyelitis without demonstrable ureteral stricture, accounts for a few of the remaining cases; chronic nephritis, evidence of recently passed "gravel" or stones and miscellaneous other causes account for a few more cases but we still have a large proportion, roughly about one-third, unaccounted for.

When we come to the study of this residual group we encounter certain rather outstanding general facts. In the first place most of these cases are in women of the hypersensitive, sometimes definitely neurotic type. General experience with this type of patient has led all diagnosticians to expect rather major pain symptoms to be produced by relatively minor pathology.

I mention this factor of hypersensitiveness or hypersusceptibility to colic pains because I believe this type of patient should always be recognized and the pain susceptibility of the individual evaluated as a fundamental step in the diagnosis, no matter what the symptom complex.

The hypersusceptibility to pain, producing spasmodic contractions of the involuntary muscle tissue of the urinary tract, is, I believe, a

rather essential factor in the group which I wish to discuss. However, we also find in these cases a real pathology which I believe acts as an actual exciting cause for the attacks of renal pain.

The following history illustrates a typical case which has now been followed over a period of eight years and seven months:

Mrs. B., age forty-five, seen in consultation, February 11, 1913. She has always been fairly robust, but of a nervous, hypersensitive type. Seven years previously she was operated by Dr. Elting of Albany. The question of renal pain was discussed at that time but Dr. Elting could find nothing wrong with her kidneys and completed the operation by removing her left tube and ovary and appendix. Following the operation she thought she was better but she continued to have some bladder trouble and occasional pains in the left upper abdomen. She is the mother of one child. There was no history in any way suggesting pelvic infection.

During the past year she had had four very severe attacks of pain centering in the region of the left kidney. The last attack began the night before I saw her with pain over the bladder followed by suddenly developing very severe pain in the left kidney region which radiated downward along the course of the ureter. For two or three days following these attacks she had painful frequent urination. The first attack came on during the night, the second attack followed an automobile ride and the other attacks had, she thought, followed slight exertions.

General physical examination showed a well developed, well nourished woman. Skin of good color, temperature and pulse normal. Chest negative. Abdomen generally hypersensitive with marked tenderness over left kidney region and at Murphy's point posteriorly. Scar of old low median incision. Vaginal examination showed nothing noteworthy. X-ray examination of the entire urinary tract showed no evidence of stone.

Cystoscopic Examination.—At the external meatus there was a small caruncle-like area. The entire urethra appeared to be thickened and hypertrophied to vaginal palpation. Even after thorough cocainization the cystoscope was passed with difficulty and with great pain and caused some bleeding of the urethra. There was no evidence of a diffuse cystitis but the area of the trigone was markedly injected. The area of injection was most marked on the left side where it extended to the region of the left ureter which was surrounded by a well marked areola. Both right and left ureters spurted clear urine.

Because of the negative urine, the normally spurting ureters, and the fear of producing an attack of renal colic, no attempt was made to catheterize the ureters at this examination but at a subsequent examination they were easily catheterized with no evidence of obstruction or hydronephrosis.

The urine was clear, sp. gr. 1011. Slightest doubtful trace of albumin. (No albumin found on repeated subsequent examination), no sugar. Centrifuged specimen showed one cast and a few cells, mostly epithelial. No evidence of pus or active infection.

Following the cystoscopic examination she had no more attacks of renal pain until September 6, 1914, a period of nineteen months; then she had a prodromal period of bladder irritability followed by a typical attack of renal colic with lesser attacks extending over a period of about a month. Finally she consented to a cystoscopic examination, which showed a recurrence of the contracted urethra. Following this examination and ureteral dilatation to F30 with a Kollman dilator, she had relief until May, 1915, or eight months, when she felt a recurrence of the prodromal bladder irritation and returned for a urethral dilatation. Again in September, 1915, she returned to have her urethra dilated. I did not see her as a

patient again until January 11, 1917. She then reported that for sixteen months she had been free from trouble but that for the past four days she had suffered from a good deal of pain in the left kidney radiating down the ureter and accompanied by vesical symptoms. Dilatation of the urethra at this time immediately started up an attack of left renal colic but following this she remained practically free from trouble for more than two years or until the summer of 1919 when she again began to have vesical irritation followed by pain in the region of the left kidney and finally typical attacks of left renal colic. Some friend told her that repeated dilatations of the urethra would surely cause cancer of the urethra so she elected a course at a well-known Sanitarium where they made almost every known kind of a test except cystoscopic examination. She continued to have vesical irritability and repeated renal pains with occasional severe colics until she returned to me for a urethral dilatation March 11, 1920. X-rays again were negative. Urine showed nothing noteworthy. Cystoscopic examination showed a moderately contracted urethra. Slight trigonal cystitis, with some very minute ulcer-like areas in the region of the left ureteral opening. A catheter was easily passed into the left ureter but it caused much pain and immediately started up an attack of renal colic. Urine from both kidneys was sterile.

Following the dilatation of the urethra in March, 1920, she remained free from renal colic until July of last year. After a prodromal period of bladder irritation she had a typical attack of renal colic in the early part of July but with no further attacks up to the present time (April, 1922).

I have recounted the above history in some detail because it is typical of a group of cases which in my experience are relatively common and comprise a large proportion of those cases of renal pain in which definite organic obstruction or active infection of the proper urinary tract cannot be demonstrated by our modern methods of diagnosis.

We have now had a fair number of these cases under observation and in all of them the clinical picture and pathologic findings and the results of treatment have been relatively constant. Most of the cases have occurred in distinctly hypersensitive types of women. All have given a history of typical renal colic associated with bladder symptoms. In all we have found an anatomically or spasmodically contracted urethra. In many the urethra had been definitely thickened. In about half of the cases we have had to dilate the urethra under cocaine anesthesia before passing a caliber F26 cystoscope. In all cases there has been a moderate but definite trigonal cystitis with more or less involvement of the ureteral orifice on the side of the renal colic. The side involved is not necessarily constant in the same case. None of these cases have presented evidences of stone, ureteral stricture, hydroureter or hydronephrosis. The passing of the cystoscope often causes a temporary cessation of the ureteral squirt but when this nervous inhibition is suspended, the squirts resume with a normal rhythm. The ureteral catheters are easily passed, except that there is sometimes a temporary spasmodic holding of the catheter in the lower end of the ureter. The urine is as a rule nega-

tive, without evidence of colon bacillus or other forms of pyelitis. It is most interesting to correlate the symptoms and the pathology in this group of cases. The phenomena of renal colic can be produced at will by the cystoscopist and its mechanism studied by means of the x-ray and pyelography. Any stimulus which will set up a spasmodic contraction of the smooth muscle tissue of the wall of the ureter or pelvis is capable of causing renal colic. Individual patients differ very greatly as to the intensity or degree of stimulus required to produce these painful spasms. In the phlegmatic type of individual, the cystoscopist can catheterize the ureters, allow the catheters to remain in place for a considerable period of time while the urine specimens are being collected, and finally the pelvis of the kidney may be filled with sodium bromide solution for x-ray purposes with very little danger of causing an attack of renal pain. On the other hand, in the neurotic, hypersensitive type of individual, even though no lesions whatsoever can be demonstrated in the urinary tract, the cystoscopic examination, no matter how gently conducted, is very liable to be followed by a severe attack of renal colic. This fact again emphasizes the point that whenever the surgeon is called upon to determine the cause of the colic-type of pain, be it renal, uterine, intestinal or hepatic, the temperament of the individual and the relative susceptibility of the individual patient to pain-producing stimuli is a factor which ought always to be given due consideration. I believe that this hypersusceptibility is an important factor in determining the renal pains suffered by the group of patients under consideration. The actual pathologic findings are quite characteristic and constant and serve, I believe, to place these cases in a distinct group by themselves.

All of our patients belonging to this group have presented more or less evidence of a so-called trigonal cystitis with some involvement of the region of one or both ureteral orifices. In my experience the colic has practically always been on the side of the more marked involvement of the ureteral area. Urologists may differ as to the cause and nature of the so-called trigonal cystitis which may amount to little more than a definite injection of the vessels of this region. Although the general involvement of the ureteral region on the painful side has been definite in each case, this involvement has not been of the type to suggest either tuberculosis or stone. This trigonal injection may be secondary to the contracted urethra and in some way caused by the same. The fact that the painful spasms subside after dilatation of the urethra would lend support to this view.

The other pathologic finding which has been constant in my cases is a contracted or spasmodic urethra. In all cases this contracture of the urethra has been perfectly evident on attempting to pass an F26

cystoscope. In a number of cases we have had to dilate the urethra by other instruments before it was possible to pass the cystoscope at all. In about one-half of the cases the urethra when examined by vaginal palpation appears to be definitely thickened.

A working hypothesis for the correlation of symptoms and pathology in these cases may be stated as follows:

In the first place we have a patient of the hypersensitive type. She develops a slight nonspecific urethritis, certainly not an uncommon disease in women. This sets up a more or less spastic condition of the urethra and favors the development of a trigonal cystitis. When this trigonal cystitis involves the region of the ureteral orifice, this ureter is sensitized, so to speak, so that with slight stimuli it develops the ureteral spasms which are the real cause of the renal colic. It is quite possible that the renal colic starts with painful spasmodic contractures of the urethra following urination and that these contractures of the urethra are transmitted through the trigone to the ureter. At any rate, if the urethra be kept dilated, the trigonal cystitis tends to disappear and likewise the urinary irritability and the tendency to renal colic

146 Barrett Street.

BACTERIOLOGY OF FATAL SYSTEMIC INFECTIONS FOLLOWING MISCARRIAGE OR ABORTION

By Willson B. Moody, M.D., Chicago, Ill.

From The John McCormick Institute for Infectious Diseases and the Pathological Laboratory of the Cook County Hospital

THIS report is based on the bacteriologic examination of fluids and organs removed at autopsy of 28 patients dying in the Cook County Hospital of systemic infection following miscarriage or abortion. It covers the period between May, 1920, and January, 1922, and is part of a study of the pathogenesis and pathologic anatomy of sepsis resulting from miscarriage or abortion, spontaneous or criminally induced. Rather extensive study has been made of the bacteriology of puerperal sepsis but it is only during the past ten or twelve years that much attention has been paid to the bacteria found in patients with evidence of systemic infection following miscarriage or abortion, and nearly all such investigations have been of the relation of the bacteria found to the prognosis and treatment rather than to the pathologic anatomy.

Schottmüller,[1] in 1920, made a study of the cervical flora of 100 cases of septic abortion and isolated in largest numbers such organisms as S. erysipelatus, staphylococcus, B. coli and pneumococcus. Winter[2] the following year reported similar results, determining the course of treatment by the bacteria isolated. Numerous papers continued this discussion concerning the bacteriology and treatment and it seems that the majority of investigators were unable to correlate the clinical course with the bacteria found in the cervical secretions. It does appear, however, that the hemolytic streptococcus is not commonly present in the cervical or uterine secretions and also that when systemic infection following abortion or miscarriage due to this organism occurs, the course is shorter and the mortality higher. Traugott[3] includes in his study of the bacteria of the lochia in septic abortion, the results in six deaths. Streptococcus longus was isolated three times, B. coli and a diplostreptococcus twice, and staphylococcus albus and a diplostreptococcus once. Thrombophlebitis, infarction and endo- or parametritis were found in those instances in which S. longus was the predominating organism; with infections due to colon bacilli and staphylococcus there was more suppuration: pelvic peritonitis, purulent parametritis and peritonitis, being noted. Warnekros[4] in seven deaths from sepsis following abortion found streptococci in the peritoneal fluid or heart blood in nearly every instance, though, in

three there was a mixed infection. Thrombophlebitis was present in three, and S. longus isolated each time. In ten cases there was one with peritonitis, four with peritonitis and sepsis, five with sepsis and pyemia. Hemolytic streptococci were found in the peritoneal fluid four times and staphylococcus once. Warnekros found that the organisms isolated from the blood were often different from those in the lochia.

Material for culture was collected by Dr. E. R. LeCount at the postmortem examination of patients dead from sepsis following miscarriage or abortion, 28 cases in all. In nearly all, cultures of the blood from the heart, of peritoneal, pleural, pericardial and cerebrospinal fluids, splenic pulp and uterine contents were made; and in all, stained direct smears were made. Blood agar plates for aërobic and anaërobic bacteria were made, and dextrose broth was also inoculated.

Hemolytic streptococci were isolated as the predominating organism in thirteen instances; B. coli in four, staphylococcus in four, pneumococcus in three, B. proteus in two, B. mucosus once, and alpha streptococci once. Mixed infections were encountered in various fluids, but the bacteria mentioned were found in each case in several of the materials examined.

Blood from the heart was cultured 27 times with positive results in 22 instances; hemolytic streptococci 17 times, B. coli and staphylococcus four each, pneumococcus, proteus, pyocyaneous and alpha streptococcus each once.

Cultures of fluid from the peritoneal cavity remained sterile in five of 21 instances. Beta streptococcus was isolated in eight instances, pneumococcus, B. coli, staphylococcus each twice, alpha streptococcus and B. mucosus each once.

It is of interest to note that twice, large numbers of gram-positive cocci were seen in cover glass preparations of peritoneal fluid but repeated cultures in solid and fluid mediums failed to yield any such organisms.

Six of 23 cultures of spinal fluid remained sterile. Beta streptococcus was isolated in five instances; staphylococcus in six; pneumococcus and B. coli twice; pyocyaneous and B. mucosus each once.

Fluid from the pleural cavities was cultured in 20 instances, yielding beta streptococcus ten times; B. coli and staphylococcus each three; pneumococcus twice; proteus and alpha streptococcus once.

From 20 cultures of the spleen, nine remained sterile; five contained beta streptococcus; two B. coli; pneumococcus, staphylococcus, proteus, and alpha streptococcus each once.

Material from the cavity of the uterus was used for cultures in 14 instances; from two nothing grew, six contained beta streptococcus,

two each B. coli and staphylococcus; proteus and alpha streptococcus each once.

Although in many cases more than one organism was recovered from the material, from the majority some one type of bacteria was found in several of the fluids examined. Summarizing the results of the 28 cases, there were 24 in which some one organism appeared in several of the fluids from that body; thus beta streptococci were isolated from more than one place in 13, pneumococcus, B. coli and staphylococcus in three each, B. mucosus and alpha streptococcus each in one. Cultures during life were made from the blood of five patients. In only one were any organisms found; these were beta streptococci and in this case there was thrombo-ulcerative endocarditis. The hemolytic streptococci isolated in all instances proved to be of the beta type; all fermented lactose and salicin but not mannite or inulin and efforts to determine any specific strain by agglutination tests failed. Agglutination of suspensions of these strains by serum from rabbits injected with strains of hemolytic streptococcus from several sources such as scarlet fever, erysipelas and infected tonsils as well as post-abortion sepsis failed to show any specific characteristic group.

Although it was noted at the postmortem that the alterations were, as a rule, peritonitis or thrombophlebitis of varying locations, degrees and sequences—also that these two sorts of alterations were rarely combined—a difference in the bacteriology to correspond to the two types of alterations was not definitely established.

Naturally enough, statements, particularly in the negative, by the patients who have suffered criminal or self-induced abortion, are entirely unreliable. Admissions were obtained from 13 patients that pregnancy was interrupted by midwife, physician, or the patient herself. In four patients, hemolytic streptococci were found; in seven staphylococcus or B. coli; and pneumococcus and proteus each once. In three of the four cases in which beta streptococci were found, it was stated that a physician or midwife had introduced rubber balloons or performed curettage. In the fourth, the patient stated that she had inserted a slippery elm stick, but it was later found that the uterus was perforated. Patients with sepsis associated with such organisms as B. coli or staphylococci or B. proteus were apparently treated under conditions far from aseptic and with repeated introductions of instruments into the uterus.

One patient aborted four days before entrance to the hospital and lived only two days after admission. At the necropsy, gangrenous endometritis and acute serofibrinous peritonitis were found, also bilateral pleuritis and plyosalpinx In the blood from the heart, peritoneal, pericardial, pleural and cerebrospinal fluids there was a pure culture of hemolytic streptococci.

Two patients denied any instrumentation of the uterus but stated that they had taken pills and used repeated douches to terminate pregnancy. Both were ill about three weeks with cramps, chills and fever. At the necropsy, gangrenous endometritis, fibrinopurulent peritonitis and perimetritis were found. In one instance there was in addition suppurative thrombophlebitis of the right iliac vein, with infarcts in the spleen and in one lung. Cultures of the blood, pleural and peritoneal fluids contained beta streptococci.

Another patient had been attended by a midwife and doctor on a number of occasions, later developing chills, fever, sweats and swelling of the left leg, with beginning gangrene. After death a purulent endometritis with an abscess in the myometrium was found, with thrombosis of the uterine, ovarian, renal, iliac, and femoral veins and inferior vena cava. Staphylococci and B. coli were isolated from the blood in the heart and the inferior vena cava. No organisms were isolated from the peritoneal fluid.

SUMMARY

From this study of the bacteria found in women dying from sepsis following abortion, it appears that the hemolytic streptococcus is more often found than other organisms, but that such bacteria as B. coli and staphylococci are not infrequently present. It is possible that the hemolytic streptococci are present in the lochia but it appears that some outside influence such as trauma from instrumentation, douches, and the like are necessary to bring about generalized infection.

REFERENCES

(1) München. med. Wchnschr., 1910, lvii, 1817. (2) Zentralbl. f. Gynäk., 1911, xxxv, 569. (3) Ztschr. f. Geburtsh. u. Gynäk., 1911, lxviii, 328. (4) Arch f. Gynäk., 1912, xeviii, 600.

Cook County Hospital.

REPEATED DYSTOCIA FROM FETAL ANOMALY IN SUCCESSIVE PREGNANCIES

By A. L. McDonald, M.D., Duluth, Minn.

THE following experience with complete *situs transversus* accompanied by polydactylism, tremendous fetal ascites and extensive anomaly of the bladder, is most unusual. Instances of *situs transversus* are not infrequent and if uncomplicated are not incompatible with life and normal growth. Repetition of almost the exact condition in the succeeding pregnancy is sufficiently unique to justify a report.

Mrs. O. F. C., age twenty-eight years. Family history: no deformity or abnormality in mother or husband, or in grandparents and families for two generations. Past history negative. Menstruation began at 14 years of age, regular 28 day type. Mother had had two pregnancies both of which resulted in premature labor. One of these babies lived but a few hours and the other was stillborn. Mother is sure that there was no external deformity of either of these babies. There is no history of previous infection or acute disease of the mother. Negative Wassermann tests had been obtained for mother and father one year previously.

Case 1.—Last menstruation May 20, 1918. The pregnancy was normal and uneventful until December 18, 1918. Patient was of normal build. General examination entirely negative. Pelvic measurements: spines 24 cm., crests 28 cm., trochanters 32 cm., external conj. 20 cm. Diagonal not reached.

Labor began at 6 P. M., December 18, 1918. Examination at 11 P. M. of that date. Abdomen was greatly distended, fetal parts could not be palpated, fetal heart heard to the right of the umbilicus. Head was not demonstrated in the fundus and diagnosis of head presentation was assumed. At 3 A. M. dilatation of cervix was nearly complete and a small part was felt protruding into the vagina. Under full anesthesia the small part was identified as a foot which was clubbed, and on which there were six (6) toes. It was apparent that we were dealing with some type of fetal anomaly. Dilatation of the cervix was completed manually, and a hand introduced into the cavity of the uterus. One could feel what was apparently a second set of membranes which were thickened but presented marked fluctuation. It was impossible to extract the fetus by traction on the foot, and careful examination demonstrated, that what was at first taken for a second bag of waters was, in reality, the tremendously distended abdomen of the fetus. A thinner area in the abdominal wall was located and the operator's finger was forced into the abdomen. A tremendous amount of clear ascitic fluid was expelled. This was not measured, but a conservative estimate would be several quarts. Extraction of the rather small fetus was then comparatively easy. Convalescence of the mother was uninterrupted. There was practically no amniotic fluid.

The baby was stillborn and its length 15 inches, the head was small and of the hydrocephalic type, thorax well formed, abdomen had been apparently immensely distended, walls thin. The lower part of the abdominal cavity and pelvis had been somewhat mutilated in the extraction, also the external genitals so that it was impossible to determine the sex of the baby. The extremities were well formed except as follows: the feet were both clubbed and on each foot six toes could be

demonstrated, the hands were normal except that there were six digits entirely formed and one of these, on each hand, was double (fused).

The liver of normal size located in the left hypochondrium, with gall bladder and ducts extending from the left side to the duodenum. Stomach, spleen and pancreas on the right side. Cecum and appendix were on the left side, only partly descended. Small and large intestines apparently normal in structure. Sigmoid and rectum to the right. In the lower abdomen there was a large mass extending from the symphysis to the umbilicus. Kidneys were of normal size and position, the ureters were enlarged and entered the mass described above. This was opened and proved to be a bladder with thickened walls. The cavity could be followed above the umbilicus, where there was a large patent urachus. On each side of the mass small structures, which were apparently the ovary and tube, were found on each side. No uterus could be demonstrated. Examination of the thorax demonstrated the heart on the right side and situs transversus apparently complete. The placenta was normal in gross structure. Section showed villi to be normal or only slightly thickened.

CASE 2.—Same mother. Last known menstruation August 20, 1920. Wassermanns: father's negative, mother's 1 plus. Mother received 3 doses of salvarsan and 10 injections of mercury. Wassermann negative. November 19, bleeding and pains, no mass or clots. Felt life in February or March. Examinations failed to demonstrate fetal motion until April or early May. Fetal heart heard in May. Fundus never extended more than two fingers above umbilicus. Duration from size seemed not more than seven months.

Labor, July 23, first stage 4 A. M., six to eight weeks beyond the expected date. Examination 5 A. M. Head engaged, occiput anterior, LOA. Fetal heart heard to left of umbilicus good. Vaginal examination: cervical dilatation ⅓. Nine A. M. fetal heart good, full dilatation. Membranes ruptured, no fluid, presenting part difficult to determine, delivery with head extended, brow presentation with the face anterior. Fetus alive but died with only a few gasps, failure of heart beat.

Examination of fetus: 16 inches from head to feet. Head fairly well formed, considerable molding, prominent frontal region, tongue split, good growth of hair. Nails not formed except at base. Thorax small but well formed. Abdomen distended with fluid. Spine and pelvis apparently normal. Extremities: uppers normal except hands. Right has seven fingers with nails, left has six. Lowers normal except feet which are clubbed, right and left with six toes each. Abdomen distended with clear fluid, about six ounces, much less than preceding case. Organs show complete *situs transversus* as follows: the liver is on the left side about normal size. Gall bladder in normal relation to liver, ducts demonstrated, common duct extends through pancreas where it receives the pancreatic duct and enters the duodenum. The pancreas extends across behind the stomach to the right. The spleen, under the right costal margin, is about three inches long and two wide. The large intestine can be followed up from the rectum with a long sigmoid flexure on the right with the descending colon attached on the right. Transverse colon in normal position, cecum and appendix on left side, descent not quite complete. The small intestine varies in size but contains bile-stained meconium. A short great omentum can be demonstrated in front of the transverse colon. Fusion is not complete as a sound can be passed through the foramen of Winslow between the layers of the great omentum. The lower abdomen is filled with a fluctuating thick walled sac which contains about six ounces of clear fluid apparently urine. Attached to one side is a small mass with a definite blood supply, later section taken to be a structure of a testicle. Opening the sac one finds a large cavity which can be followed up to the umbilicus to a patent urachus, the walls are from one-fourth to one-half inch thick. On the right side a greatly distended and tortuous ureter can

be followed to a rather small kidney which is normally located with a normal blood supply. The adrenal appears normal. A similar condition was found on the other side. The urethra can be demonstrated opening into the bladder between the ureteral openings. The external genitals are those of a male with undescended testicles. The placenta is of normal size and gross appearance.

These cases present four types of anomaly: 1st, polydactylism and syndactylism; 2nd, *situs transversus;* 3rd, some anomaly of the urinary tract with hypertrophy of the bladder wall and patent urachus; 4th, ascites. Considerations as to causal factors are purely speculative. The available literature does not give sufficient information or even suggestion to warrant detailed discussion. It is conceivable that anomalies associated with the transposition of viscera might cause the development of urethral obstruction. This in turn might explain the hypertrophy of the bladder wall, patent urachus and ureteral dilatation. I am aware of no known relationship which would explain the association of ascites, which was found in each of these cases. Polydactylism and syndactylism are known to be transmitted and are claimed to follow Mendel's Law. (J. S. Manson.[1]) This condition is easy to trace in families since it cannot be concealed and is usually observed by relatives. This author traces a Welch family through four generations. The paternal ancestor had webbing of the third and fourth fingers of each hand and six toes on each foot. His wife was normal. Sixty-four descendants are traced, of whom 27 were affected, and 33 normal, and four unknown. In most instances the affection persisted nearly true to type. It was carried through into the fifth generation, always through affected parents. There was no instance of skipping through unaffected parents. It was carried through either male or female parents. In our instance both mother and father were normal, as were their brothers and sisters. Neither mother nor father had heard of such anomaly occurring in their parents' families or among their grandparents. These two cases must be regarded as accidental or at least independent of direct hereditary influence.

The conclusions of Mall,[2, 3] based on an intensive study of a large number of pathological ova and embryos, are most interesting and have a direct bearing on the subject. The following statements are formulated from his articles. Spontaneous abortion occurs in at least 20 per cent of all pregnancies. Of all abortions, from 40 to 60 per cent are associated with pathological ova or fetuses. The abnormalities are produced during the first two months of pregnancy before development of the placenta is complete. Of such pathological ova, only one in twelve can be carried to full term and this results in a monster with varying degrees of abnormality. In tubal pregnancy the frequency of pathological ova runs as high as 96 per cent, and of

the ectopic pregnancies which persist to a period of viability, the percentage of abnormality is about as great. There is some reason to believe that a threatened spontaneous abortion during the first two months of pregnancy is evidence of a pathologic ovum which if carried to full term would result in some type of a monster. Mall is convinced that such pathologic conditions develop in normal ova from normal germ and sperm cells, due to abnormal implantation of the ovum in a defective or abnormal decidua. Corner[4] brings out the fact that abnormalities may develop in the mammalian ovum before implantation takes place. Mall in his articles describes "dissociation" of tissues, which results from disturbance in nutrition during early embryonic life, in a most interesting manner. That is, growth and development of certain tissues in certain regions are delayed at a critical period while other tissues grow, disproportionately, and the normal relationship is lost. The result is some degree of abnormality, which may be sufficient to destroy the fetus and bring on an abortion, or may be so slight that fetal development goes on to full term, resulting in some type of monster. There is a marked tendency for the same mother to produce pathologic ova or fetuses in repeated pregnancies. Pathologic ova are commonly believed to be due to abnormal conditions in the endometrium, but these are not properly associated with monsters, or abnormalities present at birth.

REFERENCES

(1) Jour. of Genetics, 1915-16, v, 51. (2) *Mall, F. P.:* Pathology of the Human Ovum. *Keibel and Mall:* Human Embryology, J. B. Lippincott Co. (3) *Mall, Franklin P.:* Am. Jour. Anatomy, 1917, xxii, No. 1. (4) *Corner, George W.:* Contributions to Embryology, No. 60. Publication No. 276 of the Carnegie Institution of Washington.

These specimens have been placed in the Anatomical Museum at the University of Minnesota, where they are available for study.

"ONCE A CESAREAN SECTION, ALWAYS A CESAREAN SECTION," AN UNTRUTH

By J. P. Greenhill, B.S., M.D., Chicago, Ill.

From the Chicago Lying-In Hospital and Dispensary

FOR the sake of statistical information, I should like to place on record very brief case reports of three patients delivered during the past year at the Chicago Lying-in Hospital, *per vias naturales*, who had in previous labors been delivered by cesarean section.

CASE REPORTS

Case 1.—Mrs. E. M., No. 10754, a primipara aged twenty, was admitted to The Chicago Lying-In Hospital, in labor, Feb. 17, 1920. The general examination was negative and the pelvis showed the following measurements: Intersp. 22, Interer. 25, Intertroch. 29, Baudeloque 18, and Conj. Diag. 11½. The antenatal record mentioned that there was a false promontory, that the lateral sacral curve was convex and that there were exostoses on the sacrum. The child was in O. L. P., the fetal heart tones were regular, the cervix was dilated 3 cm. and the head was not engaged. After twenty hours of strong pains with very little progress, a laparotrachelotomy (low cervical cesarean section) was performed, and a child weighing 3,231 gm. (7 lb. 2 oz.) was delivered. The convalescence was entirely uneventful and the patient left the hospital on the twelfth day after operation.

On May 28, 1921, this patient (No. 17376) returned to the hospital again in labor. It was decided to give her a test of labor under strict supervision. Labor progressed uneventfully and after 14½ hours there was complete dilatation and the head was on the perineum. Prophylactic forceps were applied to save the patient the strain of the second stage and a live child weighing 3,355 gm. (7 lb. 6 oz.) was delievered. After delivery the uterine cavity was carefully examined for evidences of the cesarean section scar but no trace was found. The patient made an excellent recovery and went home on the eleventh day postpartum.

Case 2.—Mrs. G. P., No. 22417, a secundipara, was admitted in labor to The Chicago Lying-In Hospital, April 30, 1922. She had had a classic cesarean section performed in August, 1920, in a local hospital, after having been in labor for twenty-four hours. The indication for the operation, according to the patient, was malposition of the baby. The latter weighed 3,855 gm. (8½ lb.). At the end of fourteen days the patient left the hospital.

At the time of the present admission, the general examination was negative. The pelvic measurements were normal, the cervix was dilated 4 cm. and the head was floating. After a labor lasting more than thirty-five hours, during most of which time the pains were strong, a baby weighing 4,025 gm. (8 lb. 14 oz.) was delivered spontaneously. No intrauterine examination was made. The puerperium was uneventful and the patient left the hospital on the tenth day.

Case 3.—Mrs. F. S., No. 11666, a primipara aged twenty-four, was admitted to The Chicago Lying-In Hospital on April 19, 1920, because she had a profuse hemorrhage from the uterus. On admission the general examination was negative. The external measurements of the pelvis were as follows: Intersp. 25, Interer. 29, In-

tertroch. 29, Baudeloque 20. The patient was at term but the head was floating. The position was O. L. P. and the fetal heart tones were regular. Vaginal examination revealed the cervix to be dilated 2 cm. and a portion of the placenta was found covering the cervical orifice. Pains began soon after admission and continued but with irregularity for thirty-eight hours. During this period there was slight bleeding. Because of this and because the head still remained above the pelvic brim, a classic cesarean section was performed. A live baby weighing 2,860 gm. (6 lb. 5 oz.) was delivered. The patient made an uneventful recovery and was discharged on the twelfth day.

On Feb. 23, 1922, the patient returned to the prenatal clinic, pregnant 26 weeks. She was advised to enter the hospital on June 2, ten days before the expected date of confinement in order to be watched. On June 9, at 2 A. M., labor began. It was decided to deliver the patient through the vagina if possible. Exactly five hours after the onset of labor, there was complete dilatation and the head was engaged. It was decided to eliminate the bearing-down pains of the second stage by applying forceps; but 20 minutes after dilatation was complete, the patient delivered spontaneously a live baby weighing 2,975 gm. (6 lb. 9 oz.). The uterus was then felt abdominally and the scar of the cesarean section was distinctly palpable. The placenta was delivered by the Schultze mechanism, by early expression, that is, by pressure on the fundus uteri after the placenta had separated and was lying in the vagina. There was no bleeding from the vagina, but examination of the placenta revealed the fact that a portion was missing. The hand was inserted into the uterus and the missing piece of placenta was found attached to the uterine scar. This piece of placenta was gently removed and the scar palpated. The thickness of the scar for most of the distance was approximately 1 cm. but in one portion it was not more than 0.5 cm. in thickness. Pituitrin and ergot were given, the former after the delivery of the baby and the latter after the expulsion of the placenta. The patient made an uneventful recovery and went home on the tenth day.

CASE 4.—Mrs. M. B., No. 24698, aged twenty-six, a tertipara, with normal pelvic measurements was delivered April 17, 1920, by a classic cesarean section in an army hospital. The baby weighed 3430 gm. (7 lb. 9oz.) and the indication for the operation was a breech presentation. Recovery was uneventful. On July 3, 1921, the patient again went into labor. She had very strong pains and according to the husband, the child's head was visible for a number of hours. Despite this, the attending physician refused to apply forceps and the patient delivered the baby spontaneously after 15 hours of hard labor. This child weighed 3628 gm. (8 lb.). For her third delivery (Sept. 21, 1922, the patient came to Dr. DeLee. She gave a history of abdominal pain suggestive of adhesions and on abdominal examination definite bands could be felt connecting the uterus with the abdominal wall. Furthermore the uterine scar was easily felt and this was considerably depressed for a distance of about 6 cm. Because of these findings and because the patient feared further pregnancies, it was decided to perform a classic cesarean section. On opening the peritoneal cavity thick connective tissue bands were found stretching from the uterus to the abdominal wall. The uterus was rotated so markedly to the left that the right cornua was located in the midline. The bands were ligated and cut, thereby releasing the torsion of the uterus. A low fundal incision was made and a live child weighing 3600 gm. (7 lb. 15 oz.) was delivered. The old scar was found to be very thin, as abdominal examination had revealed. After suturing the uterine incision, wedges from the uterine cornua were removed, the raw edges covered with peritoneum and the proximal ends of the fallopian tubes buried in the folds of the broad ligaments. The patient made an excellent recovery and went home on the fourteenth day.

COMMENT

It will be noted that in all four cases, the babies born through the vaginal route, weighed more than the children delivered by cesarean section. One patient was delivered by forceps (prophylactic) and three gave birth spontaneously. In three cases the uterine cavity was explored. In the cases where the classic cesarean section had been done, a relatively thin scar was found, whereas in the patient on whom the low cervical operation had been performed, no trace of a scar was palpable. In the three cesarean sections performed at the Chicago Lying-in Hospital catgut only was used in closing the uterus.

It appears from this report that the dictum, "Once a cesarean section, always a cesarean section," is untenable in the light of the modern way of performing cesarean section.

426 East Fifty-first Street.

Society Transactions

THE NEW YORK OBSTETRICAL SOCIETY

MEETING OF OCTOBER 10, 1922

THE PRESIDENT, DR. R. H. POMEROY, IN THE CHAIR

DR. ONSLOW A. GORDON, JR., reported a case with marked **Cystic Changes in Uterine Myomata.**

This case occurred on the Gynecological Service of Bellevue Hospital and seemed of special interest because of its well-developed cystic type and the symptomatic and gross resemblance to an ovarian cystoma.

The patient was single, forty-four years of age, her menstruation showed no ab-

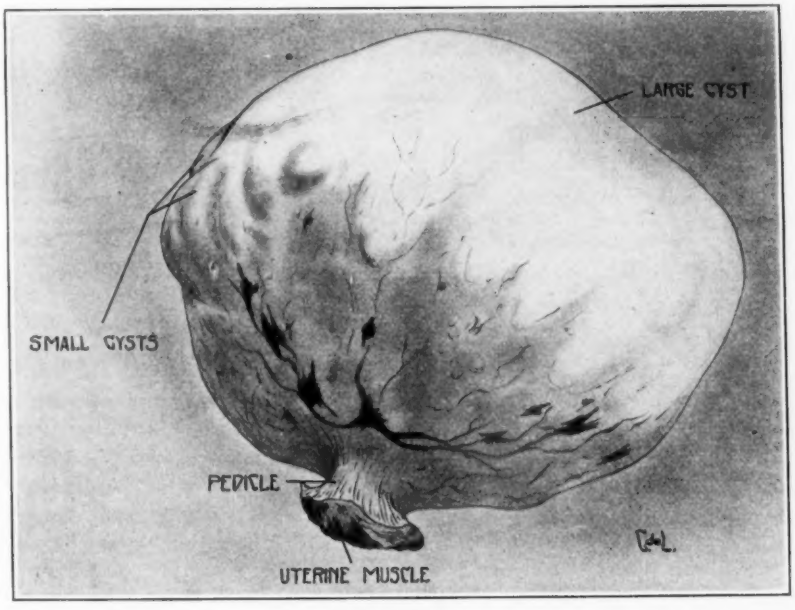

Fig. 1.

normality, occurring every twenty-eight days and lasting three days. She had never been pregnant. She had noticed an almost continual "soreness in the abdomen" for about two years; three days prior to her admission to the hospital the pain suddenly became severe and she was unable to walk. There were no gastro-intestinal symptoms.

Abdominal examination disclosed a plainly palpable tumor mass, occupying the central portion of the lower abdomen extending almost to the umbilicus and measur-

ing about 15 cm. in diameter. The mass was regular in outline, apparently cystic and not tender.

Vaginal examination showed a normal sized uterus anterior to the cystic mass described. There were no other palpable abnormalities. A tentative diagnosis of ovarian cyst or fibromyoma was made and a laparotomy advised.

When the abdomen was opened a glistening grayish colored cystic mass presented which was thought, because of its gross appearance to be an ovarian cyst. It was found, however, to arise from the fundus of the uterus just posterior to the right fallopian tube. It had, as shown in Figs. 1 and 2, a definite pedicle, which was excised.

The specimen consisted of a rounded mass measuring 15 x 15 x about 10 cm. At one pole of the mass was a small oval nodule, measuring 2 cm. in length. This nodule is composed of tissue which has all the naked eye characteristics of well-

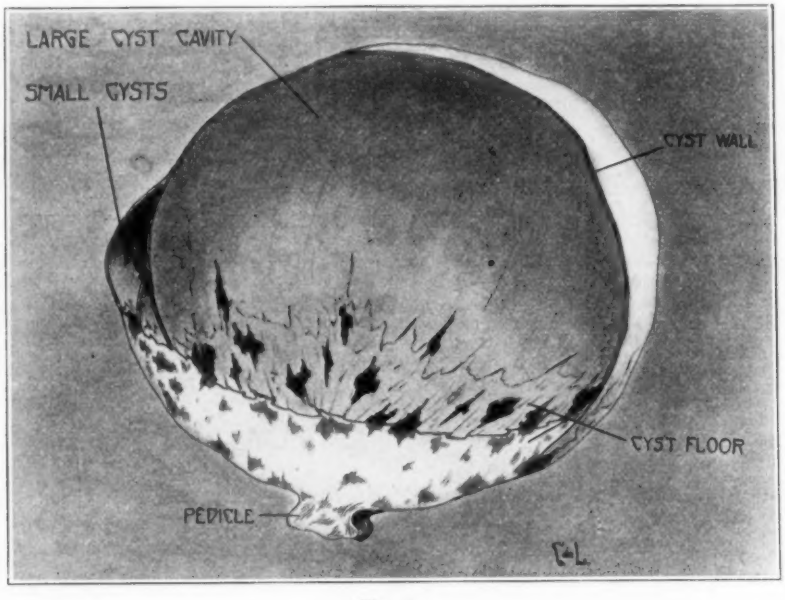

Fig. 2.

preserved uterine muscle. From this nodular pedicle a large, thick, fan-shaped mass of tissue ran off circumferentially for a distance of about 8 cm., when it suddenly became replaced by a thin rim of smooth, tough, glistening fibrous tissue, puncture of which released about 300 c.c. of thin, clear fluid.

Microscopic examination of the wall of the cyst at the point of junction with the remnants of muscle tissue shows the presence of an external layer of fibrous connective tissue, constituting the wall of the cyst-like formation. Internal to this are remnants of relatively well-preserved uterine muscle fibers, together with patches of granulation tissue, mature fibrous tissue, and variable quantities of fibrinoid material of indefinite structure.

Transudation changes, of which this cystic fibromyoma is a type, occur as a rule in well-pedunculated subserous growths permitting a moderate circulatory interference especially with the return blood supply. Areas of hyaline degeneration are formed which gradually undergo liquefaction and small cyst spaces thus develop. Such spaces have, of course, neither an epithelial nor an endothelial lining. As

these spaces enlarge the trabeculae separating neighboring spaces rupture and thus larger spaces are formed. Enormous cystic myomata may thus develop; Lihotsky reports a case in which the cyst contained 34 liters; Cullen reports a cystic myoma weighing 89 pounds. The fluid content of these cysts is usually straw-colored and if hyalinization has preceded the cyst formation it will coagulate upon exposure to the air.

DR. JOHN H. TELFAIR reported three cases of **Separation of the Symphysis Pubis during Labor.**

At a meeting of this Society in January, 1916, Morgan reported four cases of ruptured pelvis. His analysis of statistics at that time indicated that this accident occurred once in 25,000 cases. The three cases about to be described were admitted to the Obstetrical Division of Fordham Hospital during the past eleven months.

CASE 1.—Anna H., age twenty-six, para i. Family and previous personal history negative. Admitted December 19, 1921, at term, all pelvic measurements normal; presentation and position, vertex, L. O. A. First stage 62 hours, second stage 1½ hours, terminated by a high forceps delivery. During the extraction, when the head was in midpelvis, the operator felt a sudden "give" after which the extraction was easy. Baby weighed 4560 grams. There was profuse bleeding from the anterior vaginal wall and upon examination it was found that the entire anterior vaginal wall, urethra and labia were separated from the ramus pubi on the left side. After introducing a catheter into the meatus, clear urine was obtained indicating no injury to bladder. There was a separation of the pubic bones of about 3 cm. Retention catheter inserted and lacerations repaired. Six inch adhesive plaster strap was applied over the trochanters and completely surrounding the pelvis. Shock was moderate, patient responded well. Two days postpartum patient developed a profuse, purulent discharge coming from the space of Retzius. Sacroiliac joints showed no disturbance. Patient ran a mild septic convalescence. Fibrous union developed at the symphysis and the patient was discharged from the hospital in four weeks, walking without difficulty.

CASE 2.—Clementine L., age twenty-six, para iv. Admitted May 28, 1922. Had had three full-term normal deliveries. Patient at term, weighed 95 pounds, normal pelvic measurements, presentation vertex; position, L. O. A. Labor progressed rapidly with strong pains. After delivery patient showed moderate shock. Baby weighed 5280 grams. Separation of the symphysis was suspected after examination and this was confirmed by x-ray. There were no lacerations. The usual adhesive straps were applied and the patient made an uneventful recovery, leaving the hospital on the sixteenth day, walking without difficulty.

CASE 3.—Josephine N., age twenty-four, para i. Admitted June 12, 1922, having just been delivered at home by forceps. Baby weighed 4560 grams. Came in still under the influence of ether, with vagina packed to control hemorrhage. Moderate shock present, pulse 136. On June 13, packing was removed, when it was found that the anterior vaginal wall and urethra were torn from the pubic bones, the laceration extending into the prevesical space. The pubic joint was ruptured and the ends of the bones separated 10 cm. Permanent catheter was introduced and the oozing wound was packed to control hemorrhage. The pelvis was strapped in the usual manner and the pubic bones brought into apposition.

Next day temperature was 103° and pulse 140, with pain and tenderness in lower

abdomen and worked distention. Patient ran a severe septic course for three weeks and left the hospital at the end of five weeks walking without pain or difficulty.

DISCUSSION

DR. F. A. DORMAN.—In my experience this condition is a rare one. Possibly a certain number of women must come to us in a condition that makes it very easy for the symphysis to rupture. I have in mind this moment a patient who had premature twins. There was practically no difficulty in delivery or in extraction and yet there was separation of the symphysis and a lot of pain postpartum. There was no history of a movable symphysis before term. Some patients develop a movable symphysis during pregnancy which is capable of a certain amount of separation but will tighten up after delivery. A very interesting case of that sort came to my hands years ago when I was in the Post-Graduate Dispensary. This particular patient limped into the examining room, exceedingly crippled. It was her third pregnancy. She said that every time she became pregnant the symphysis softened and as soon as the baby was delivered and she had rested for a time everything tightened up. We must have graduations of that sort in this condition, and a certain number of our cases, even with a slight amount of trauma, will, I believe separate. I would like to ask the doctor whether there was sacroiliac pain in his cases?

DR. J. O. POLAK.—These separations are not so infrequent in the emergency services. I have only seen four, but three of the four occurred with spontaneous labor, and the fourth one occurred from just such an incidence as Dr. Telfair reported, where high forceps were used.

Fortunately, in Brooklyn, we have been able to teach that forceps are not to be applied on floating heads, and so in the last five or ten years I have not seen any separations of the symphysis. A remarkable instance was that of a case in the service of Dr. Beach where the patient had a separation, prior to delivery, of five or six centimeters, but was able to walk without difficulty. The separation increased during delivery, but she was up in the usual time. The separation still exists.

DR. A. H. MORSE.—We had last year a case similar to one of those Dr. Telfair described, a patient who had been delivered outside the hospital. We were unable to get an accurate history, but there had been, at any rate, a forceps delivery and she was sent into the hospital in shock and with a separation of the symphysis so that one could introduce three fingers. There were also in that case two lacerations in the bladder. We strapped her in much the same way that Dr. Telfair described and followed the case up with the result that after having x-ray pictures taken we found that there was a definite injury at the symphysis. She could get about, but she passed out of our hands and later we discovered that she was operated on, but without success.

I have seen one other case in which forceps was attempted in a moderately contracted pelvis in a colored woman and during the traction the symphysis separated.

DR. O. P. HUMPSTONE.—I would like to refer to postpartum separation of the symphysis. I had never seen this condition before, and it seems to have some points of interest. This woman was brought in profoundly septic from her labor outside the hospital, and there was no separation of her symphysis at that time. She developed a parametritic exudate (extraperitoneal) pointing towards Poupart's ligament on the right side and ultimately opening in the left labium. In two or three days, however, there was a swelling in the right labium. A small incision permitted the discharge of about a quart of pus. Incising and exploring

up along this tract, we found that the patient's symphysis was gone and that the cartilage had been entirely destroyed by this septic process, but ultimately she made a perfectly good recovery and was able to walk.

DR. TELFAIR, closing.—In answer to Dr. Dorman's question as to whether there were any symptoms or evidence of sacroiliac pain or separation, I might say that the plates of the last case I described showed a considerable separation of the sacroiliac joint on each side; but none of these patients had any pain as a result of sacroiliac separation.

I might say, incidentally, that it is a very difficult matter to actually hold these cases of separation in apposition; in fact, one, I think, should be careful not to seek complete apposition on account of the great danger of squeezing the urethra, the anterior vaginal wall and the base of the bladder between the ends of the bones.

When case No. 3 left the hospital, I had the opportunity of examining her in the standing position, shifting the weight of the body from one foot to the other, and fibrous union was so perfectly developed that one could easily notice the sides of the pelvis rock up and down; it was a very satisfactory joint and the patient had no pain when walking.

DR. ROBERT T. FRANK (by invitation) presented a paper entitled **Treatment of Cystocele, Rectocele, and Uterine Prolapse.** (For original article see page 8.)

DISCUSSION

DR. L. G. BALDWIN.—Insofar as operations for repair during a woman's childbearing period are concerned, there are very few cases that cannot be made comfortable, and I think by all means they should be done. By the use of pessaries or the use of the cautery in healing erosions, stopping discharge, and doing away with sepsis, women can be made perfectly comfortable.

I think there is nothing more outrageous than the present-day operations on the cervix and perineum after every childbirth.

DR. H. N. VINEBERG.—Dr. Frank has given us a very lucid description of original work, especially insofar as the amount of cellular tissue surrounding the cervix is concerned and the importance of conserving it. I have tried in these cases, where there is a prolapse and the uterus is enlarged, to amputate the body of the uterus and keep the cervix and cellular tissue around it and use it as a prop for the bladder and for the anterior portion of the pelvic floor. I would like to ask whether there is any advantage in separating the vaginal mucosa from the fascia? Cannot the same result be obtained by simply suturing the two together instead of doing a very difficult separation? I believe you get the same result if you are sure that the vaginal mucosa is denuded far enough out so that the fascia is underneath. The fascia near the pubic arch is very thick and strong and I always suture this separately. The fascia lower down very often is thin and to suture this separately from the vaginal mucosa, in my opinion, is not a wise procedure. I did it in a few instances and I regretted it.

I am also gratified to find that Dr. Frank has found by experience that exposing the levator ani muscles and suturing them together has not been satisfactory. My previous experience in these cases of rectocele was that while I obtained a pretty good lower third of the vagina, some of the rectocele would come over and prolapse on the shelf which was built up below. Now, I simply

carry my denudation higher up, almost to the cervix posteriorly, making a wide straight denudation and pass the sutures so as to embrace the fascia externally, coming out in the opposite side at the point of entrance. The patients are comfortable after this procedure. I wish I could say as much for the cystoceles as I can for the rectoceles. I seldom have a recurrence of a rectocele, but there are cases where the whole anterior vaginal wall comes down and there is practically no fascia. I do not know what one can do to keep up the bladder completely in those cases.

DR. A. M. JUDD.—I want to add my word of approval to the remark of Dr. Frank that he still uses the Alexander, or the Alexander-Adams operation. It is an operation which I have done for a great many years as an aid in plastic work and it gives me just as much, or better, success than when I first began to use it.

I also want to add my word of commendation of the doctor when he condemns ventral suspension as a curative measure directed towards the relief of uterine prolapse.

In closing I would like to say that I have been doing the Sims-Emmett-Baldwin operation for the last twelve years and it gives me absolute satisfaction.

DR. H. C. TAYLOR.—I think that everyone who has read Dr. Frank's articles will appreciate the thoroughness with which he has gone into the subject.

Let us consider first women who may have more children. I do not sew up every perineum and every cervix that has been torn, but there is no question that some women in the childbearing age who have lacerations which should be repaired, as otherwise they will get distinctly worse and when they are finally repaired it will be more difficult of accomplishment. So I would be very far from throwing out all cases of women who are going to have more children.

Let us consider next only those women who are beyond the childbearing age. There are five different structures that can be displaced and must be kept in mind for the sake of clearness. They are the urethra, the anterior vaginal wall, the posterior vaginal wall above the perineum, the uterus and the perineum. There may be any one or all, or any combination of these structures in the displacement.

With a displacement of the anterior vaginal wall of any extent the operation I do is the replacement of the bladder to a position corresponding more or less to its original position (it is doubtful that we ever replace it exactly) with a suturing of the anterior wall of the vagina to the anterior surface of the uterus. With a senile uterus and a marked cystocele the bladder is displaced as high as the top of the uterus and we have a true interposition operation. If the uterus is large and the displacement of the anterior vaginal wall is only moderate it is not necessary to displace the bladder so far and the operation is not strictly an interposition operation though the difference is one of degree and not kind.

It is my opinion that the expression "interposition operation" is an unfortunate one as the operation is really replacement of the bladder and is only an interposition operation when the uterus is small and the prolapse marked.

So far as a hysterectomy for prolapse of the uterus or vaginal walls is concerned I rarely do one unless there are other conditions, such as a tumor in the fundus of the uterus, that require the hysterectomy. With a normal uterus a hysterectomy is rarely indicated.

I frequently do a ventrofixation for a prolapse of the uterus in addition to the plastic work on the vagina in women beyond the childbearing age and it has given good satisfaction in my experience. I do not mean a ventrosuspension nor any form of round ligament suspension but a firm fixation of the fundus of the uterus to the abdominal wall.

Regarding the treatment of rectocele, many talk of obliterating the culdesac. I sometimes open the culdesac but I do not obliterate it, nor can I see how the other men do it. I always begin my operation high in rectoceles in women beyond the childbearing age, in practically every case the denudation begins at the cervix. This gives a firmer and more satisfactory support of the pelvic floor.

DR. DOUGAL BISSELL.—Fifteen years ago I made a practical study of the intraabdominal fascia and its many ramifications, and secured it in its entirety, so that it could be demonstrated as one continuous fascia. The dissection was begun at the upper abdominal diaphragm and followed down to the lower or pelvic diaphragm, including the suprarenal glands, kidneys, ureters and female genital organs. My object was not only to demonstrate the unity of the fascia, but to acquire a better understanding of nature's design in the mechanical supports of the female genital organs. The knowledge acquired in this study convinced me that while these several fascias of the female pelvis were ramifications of a continuous fascial structure, each performed separate or special functions, and that rectocele and cystocele, the result of injury or over stretching, were separate entities.

It is gratifying to know that Dr. Frank has abandoned the technic of levator ani muscle union when repairing the lacerated perineum. I have persistently condemned it as an illogical, unanatomical and unscientific procedure. If the injured perineal body is properly restored, provided the posterior vaginal wall is not injured, the levator ani will approximate and function normally without being attacked surgically. The function of the perineum is limited to the anal region. If an injury exists which results in an abnormal separation of the levator muscles resulting in a protrusion of the posterior vaginal wall, these muscles should not be the tissues directly treated, but the relaxed rectal or musculofascial structure of the posterior vaginal wall, and this structure is connected with the levator muscle in such a manner as to make the muscles approach each other when properly readjusted.

Destruction of the perineum may be partial or complete, without an associated rectocele and *vice versa* an extensive rectocele may exist without an associated injury to the perineum. The anterior barrier of the rectum is not the perineum, but the rectal or musculofascial structure of the posterior vaginal wall. It is the failure to recognize the independent function of these areas that has caused a confusion regarding the etiology and surgical treatment of injuries to the posterior genital tract.

My experience agrees with that of Dr. Howard Taylor with regard to enterocele, to separate a large portion of the peritoneum in Douglas' culdesac and tie off where an enterocele exists, does not obliterate the culdesac. I am in accord with him in the opinion that the culdesac is not, under these circumstances, obliterated as is commonly supposed. My experience with the results of such surgery, per abdomen, by the advocates of this operation, has convinced me that the remedy is worse than the disease.

DR. JOSEPH BRETTAUER.—After all, the various speakers do not differ very materially; it is simply a matter of describing the same tissue by different names. The principle of uniting the torn pelvic fascia is the outstanding feature in the various methods. We have never attempted a direct suture of the torn levator muscle and have seen very unsatisfactory results arising from this procedure. The results of the methods described by Dr. Frank are in the main satisfactory; however, the degree of relaxation and prolapse of the vagnal walls varies so much, that of necessity, the extent of the operation must be adapted to each individual case.

Some operators have not been able to obliterate the Douglas pouch by interfer-

ence from below. In those extreme cases in which we find an enterocele in addition to a pronounced rectocele, we have always divided the operations into two parts; first, the one which closes the Douglas pouch by circular seroserous sutures, and second, an extensive posterior colpoperineorrhaphy.

Surely we all agree with Dr. Frank that we cannot be too emphatic in declaring that women during the childbearing age should not be subjected to indiscriminate operations. Indication for operation should be found only after the most careful consideration.

DR. FRANK (closing).—Insofar as the technic is concerned, I also prefaced my remarks by saying I had no intention of trenching upon the delicate subject of priority. In 1870, 1880 and 1890, the basic principles were understood, but they were not clarified. Dr. Brettauer, when I first came to Mount Sinai Hospital, about 1900, could do very excellent plastic operations, as good as any one of us can do now, but he could not teach me how to do them, and it was for that reason that I went into the anatomical laboratory and worked just along the lines Dr Bissell has indicated, in order to see what supports could be utilized and how I could expose them so as to utilize them. I might say that although we differ in minutiae of technic, I think we are now working along the same line, because we use the fascial structures and are most careful in leaving the muscles unexposed. and only approximate them together with their fascia.

Dr. Vineberg spoke about separating or not separating the vaginal mucosa from the pubocervical fascia. I think it is a distinct advantage to do so, and I always say that the only way to expose the anterior fascial structures correctly is to make an incision to only a very slight depth, and then use blunt dissection. It is a bloodless operation and you expose the fascial structures well. You can reach out as far as the parametrium in some cases.

As far as exposing the levator ani muscle is concerned, I assure you that in many of the operations that I have witnessed it is not that muscle that is exposed but the deep perineus muscle. Again and again I have seen the perineus profundus pointed out as the levator.

I did not speak of ventral suspension at all, because I think it is not a good operation; but I agree fully with Dr. Taylor that, after the menopause or after sterilization, there is no better operation than a firm low ventral fixation. The bad results are very few.

As far as Dr. Taylor's modifying the indications that I laid down, I would say that I fully agree that if we watch a case and see a progression of a prolapse, for example, it is time to interfere; but what I take issue, not with Dr. Taylor, however, but with many members of the profession, is in advising the patient at the first visit to have a slight or moderate perineal injury repaired. With that view I am sure he will agree.

As far as lacerations of the cervix are concerned, I believe that only such cases which are apparently causing trouble should be operated upon—the troubles being repeated abortion, and a profuse leucorrheal discharge.

Perhaps Dr. Taylor and I will not be so far apart if I modify my nomenclature and call obliteration of Douglas' culdesac simply a shortening of the culdesac. I do not claim for a moment that you can completely obliterate the culdesac from below. With the Moschcowitz operation you can shorten it appreciably from above and prevent any prolapse of the intestines behind the cervix and the posterior vaginal wall.

Dr. Bissell emphasizes that it is the fascial structures which lend support to the uterus above the anterior and posterior vaginal wall. The vagina as it goes through the levator gap (the genital hiatus or anterior edge of the levators) re-

ceives fascial investments which go up and downward. Beyond the levators it receives additional investment from both layers of the triangular ligament.

I did not go into the minutiae of the anatomy describing how the abdominal fascia also gives investment to the upper portion of the vagina. The muscular portion of the pelvis is simply an additional safeguard which resists sudden stresses and closes up the gap during coughing or expulsive efforts.

I agree with Dr. Brettauer that there are certain cases that cannot be cured permanently by any method, and this certainly applies to rare cases of cystocele where there is no fascia left. In my paper I stated that to be the case. In other words, we are in the same identical position with these cases that we are in in ordinary herniae, whether umbilical, inguinal or other. You may expect 5 to 10 per cent of recurrences. If any one claims he can cure every case of pelvic injury, either uterine prolapse or the associated conditions, I simply say that he is either not a sharp critic of himself or that he does not see his cases after operation.

ST. LOUIS GYNECOLOGICAL SOCIETY

REGULAR MEETING, APRIL 14, 1922

DR. H. S. CROSSEN, IN THE CHAIR

DRS. F. P. MCNALLEY AND W. J. DIECKMANN read a paper on the **Hemorrhagic Lesions of the Placenta and Their Relation to White Infarct Formation.** (For original article see page 55.)

ABSTRACT OF DISCUSSION

DR. MCNALLEY (closing).—Dr. Schmitz's diagnosis of an infarct in his case might fit in very well with Young's idea of infarct formation. The recurring pain in pregnancy in his patient may be caused by small retroplacental hemorrhages and thus an infarct may finally result. If such is the case she should develop a toxemia. It will be interesting to follow her course and examine the placenta after her delivery.

Dr. Gellhorn asks whether every white infarct has its origin in a hemorrhagic lesion. We do not know whether this is so. If Williams' idea is correct, it is not so. We are convinced that some have their origin in red infarcts as described by Young and that some have their origin as hematomata.

We have noticed no particular difference in the size of the villi in toxemic and other cases except in syphilis where, of course, they are much larger.

Young says interference with the maternal circulation will cause circumscribed infarcts because different portions of the placenta are supplied by different vessels and that the whole placental site is not merely a huge lake of blood with free intercommunication of all parts.

One of the explanations that has been advanced for infarct formation is that it is a senile change in the placenta. If this is accepted it will account for the frequency and degree of infarct formation found in those cases that have gone overtime.

In regard to Dr. Ehrenfest's discussion: It is Young's idea that absorption of toxins from infarcts is the cause of the toxemia. The infarcts are the result of interference with maternal circulation, i.e., the changes in the maternal vessels are primary, the cause and not the result of the toxemia.

We agree with Dr. Otto Schwarz that these groups of cells are probably decidual rather than trophoblastic and have noticed their association with white infarct formation. We do not accept his suggestion that the hematomata in the placenta could be accounted for by rupture of a maternal vessel because there are no vessels at the site of these lesions.

In answer to Dr. Taussig's question, we feel that it is possible to differentiate microscopically between a white infarct formed from a red infarct and one formed from a hematoma. In the former the outlines of some villi may be seen, while in the latter these are absent.

We have made no attempt to determine the relation of the weight of all the infarcts to the weight of the entire placenta. There is a specimen in the laboratory that is at least three-fourths infarct.

DRS. O. S. KREBS AND A. P. BRIGGS read a paper entitled **Blood Studies in Normal Pregnancies.** (For original article see page 67.)

ABSTRACT OF DISCUSSION

DR. KREBS (closing).—In answer to Dr. Taussig's question, we might say that the result of chemical studies on the blood of pregnant women, to the present time, have thrown little light on the possible etiology of the toxemias. The nitrogenous elements have been thoroughly investigated, but the inorganic constituents, with a few exceptions, have been neglected. For this reason we undertook a study of the latter in the blood of normal pregnant women, hoping to arrive at normal values for comparison with future work in abnormal cases. Our results have shown the constancy of the inorganic salts in the blood of normally pregnant women, regardless of the period of gestation, except that in the latter weeks the calcium content is slightly diminished.

As Dr. Gellhorn has emphasized and as has been brought out by Caldwell and Lyle, a given blood picture is only of significance together with the clinical findings at the time the blood sample was taken. Dr. Gellhorn suggests that cases should be taken in hand before retinitis develops, to which everyone agrees. But given a pregnant woman with retinitis complicating a toxemia, then the problem arises to determine the amount of kidney damage, especially since the advisability of future pregnancies depends to a considerable extent upon this diagnosis.

Dr. Ehrenfest correctly emphasizes the value of the results of determination of the nitrogenous elements in the blood in relation to the prognosis and advisability of future pregnancies. We feel that the results must be interpreted in terms of renal insufficiency, and, too, that blood studies as have been carried on in the past are of considerable value in the differential diagnosis and prognosis of the various toxemias, but that they throw no light on the exact nature of the toxemia or the probable source of the toxins.

Dr. Royston emphasizes the importance of the estimation of the twenty-four hour urinary output, and a diminution of this as a danger signal. Williams in his text book, speaking of pre-eclamptic toxemia, states that it should be suspected when the patient complains of headache, lassitude and various of the other usual symptoms, but particularly if the urine is diminished in amount and contains albumin.

Department of Reviews and Abstracts

Conducted by Hugo Ehrenfest, M.D., Associate Editor

Collective Review

Midwifery in India*

By Kedarnath Das, M.D., Calcutta, India

THE origin of the ancient medical literature of India can be traced to Ayurveda, the date of which is supposed to be about 1500 B.C. The most renowned representatives of this literary monument are the ancient trio—Charaka, Susruta and Bagbhata. Charaka probably lived about the commencement of the Christian era, Susruta during the fifth and Bagbhata, not later than the seventh century, A.D. In tracing the progress of medicine, it is necessary to observe that human nature exhibits a general resemblance among all nations which, however erroneous, must be acknowledged to have some foundation on facts. In spite of the supposed exclusiveness of Egyptian culture, there appears to be an undoubted spasmodic fecundation from Hindustan. It is an interesting fact that the ancient civilized races—Egyptians and Hindus—endowed the art of healing with a supernatural origin and brought a number of deities in close relationship with medicine. Among others, the Egyptians worshiped Bubastis, the goddess with a cat's head as the tutelary deity of procreation and childbirth, while the Hindus worship Sasti, the feline goddess of maternity and the protectress of children, who rides on a cat. The ancient Sanskrit literature, however, enables us not only to trace the development of midwifery in India from its primeval origin in empiricism and theurgy to its height as a fairly completed, though empirical, system of learning, but also to show the determining influence which the East exercised on the trend of medical thought of Greece.

I shall now make a few observations on the salient points on the subject of midwifery, which can be gathered from the writings of ancient Hindu physicians. I might tell you that there was no separate or special treatise on midwifery but scattered references to obstetric subjects could be found in the medical works. Of the ancient trio, Susruta is characterized by a fuller consideration of surgery and difficult labors. It is noteworthy that in their surgical technic, the Hindus were distinguished above all their brethren of the neighboring oriental countries. This superiority was due to their scrupulous cleanliness and the minute attention which they paid to details.

The anatomical descriptions of the pelvis and female organs of gen-

*Read before the Forty-seventh Annual Meeting of the American Gynecological Society, May 1-3, 1922, at Washington, D. C.

eration are far from accurate and often fantastic, drawn from imagination, rather than from observation. The same remark applies to their knowledge of the physiology of reproduction but the clinical observations regarding the age of onset of menstruation, the nubile age and the most favorable time for conception, are worthy of note. The menarche is said to occur at twelve, which I may point out, corresponds to the observations made by me and published in Davidson's *Hygiene and Diseases of Warm Climates*, in 1893. These were undertaken to ascertain the influence of climate on menstruation. I may also remark, that the practice of early marriage, which was in vogue in Bengal, but which is gradually dying out, was not sanctioned by ancient medical writers but was brought about by social customs. Maternal impression on the fetus is pointedly referred to. The hygiene of menstruation receives adequate treatment.

THE GROWTH AND DEVELOPMENT OF THE EMBRYO

This subject was studied in great detail, but here, too, imagination played a great part. The question of controlling the sex of the child is discussed at great length and detailed directions are given as to the proper time of sexual conjugation, diet, dress and mental conditions, if a male offspring is desired.

The Signs and Symptoms of Pregnancy.—The following only are mentioned: pigmentation of areola of the breast, hypertrichiasis, salivation, sickness and vomiting. In addition to these, weakness and heaviness of eyelids are mentioned. Regarding differential diagnosis of pregnancy, it is pointed out that there is a kind of gulma (tumor) which simulates pregnancy. If after ten months of a supposed pregnancy a child is not delivered, the condition must be considered abnormal and suitable treatment should be adopted.

Hygiene and Management of Pregnancy.—Coitus during pregnancy is interdicted. Rest and quiet with a free and easy life were enjoined. The whims and caprices of the mother should be satisfied; otherwise the child will be born with some defect or deformity. Details of diet in the various months are given.

Duration of Pregnancy.—Nine calendar months seem necessary for the perfection of the fetus; that is 39 weeks or *273 days* from conception. The Hindu sastras allow 10 calendar months or 300 days.

Abortion.—This subject is treated with great detail, especially with reference to its therapy, both preventive and curative. A large number of drugs have been recommended. There is a clear reference to cases of missed abortion, in which exercise is recommended, evidently with a view to expedite the expulsion of the fetus. There is also a mention of the removal of the fruit of conception by the influence of devils. An elaborate list of diets in different months is given so as to prevent abortion. A distinction is made between abortion in the first four months and abortion after that period.

Diseases of Pregnancy—are very meagerly dealt with. A form of nervous disease, complicated with convulsions is described. This is said to have been uniformly fatal, perhaps tetanus.

Diseases and Abnormalities of the Fetus.—The only thing mentioned regarding their causation is the influence of disease in parents.

Abdominal inspection as a diagnostic method during pregnancy was

evidently in use. It is said that twins are known by a depression along the centre of the abdomen.

LABOR

Preparation for Labor.—The description of the lying-in chamber is given very minutely indeed. The directions laid down are undoubtedly rational and hygienic. They show that preventive midwifery made great progress and successfully too. A comparison of the descriptions as given by ancient writers with those of the modern physicians tend to show how custom and usage have rendered the management of labor in some of the Hindu houses of today positively dangerous.

The Fetus in Utero.—With regard to the presentation, position or lie of the fetus, the ancient had absolutely no knowledge. It is merely mentioned that the fetus lies in the uterus bent on itself with its hands and feet flexed.

Physiology of Labor.—Knowledge under this heading was absolutely nil. The explanation of the method of expulsion of the child was grotesque.

Management of Labor.—The signs of the approach of labor are given fairly accurately. During labor, patient lies on her back with high pillows under her head, thighs separated and legs bent. Four old experienced women attend; some directions are given for their guidance, but there is nothing to show that any attention was given to the care of the perineum. It is mentioned that "should the child advance crooked it is to be turned straight by manual assistance." I cannot explain to what it refers. To expedite the expulsion of the placenta, various methods (some disgusting) were adopted to induce vomiting.

Management of Puerperium.—The directions given are far from satisfactory, as they are based on an erroneous conception of the normal physiological course of the puerperium.

Multiple Pregnancy.—Nothing was known as regards its causation beyond the mistaken idea of deranged air.

Delayed Labor.—Drugs for promoting the expulsion of the fetus were not known. Fumigations, charms and sometimes manipulations were employed to expedite delivery.

Preternatural Labor.—There is no attempt at classification, but several conditions which produce dystocia and require artificial aid, are described. Susruta describes eight abnormal presentations, of which one, (both lower extremities), does not require any assistance, five require assistance with the hands alone (some kind of version or extraction) and two instrumental interference (i. e., embryotomy). Destructive operations on a living child are not recommended.

Of other complications, unconsciousness and nervous diseases are referred to, which are said to be always dangerous. Perhaps eclampsia is mentioned. Contraction or any disease of the vagina (? passages), is always considered to produce difficult and dangerous parturition. Death of the fetus in utero connoted great danger to the mother and immediate removal of the child is recommended. Signs of death of the fetus are described: cessation of the child's movements and fetid smell of the breath being mentioned. It is noteworthy that hemorrhage, ante- or postpartum is not even touched upon.

Obstetric Operations.—Postmortem cesarean section is mentioned. Perforation of the head, followed by cranioclasm and extraction of the

fetus by hooks is described. Evisceration is recommended for swelling of the fetal abdomen. In impacted breech, division of the pelvic bones is resorted to. The after-treatment of these cases receives careful consideration.

Obstetric Instruments.—Only a few are described but the description of these instruments is neither minute nor precise, as they are not illustrated by drawings nor now employed. In general the name of the instrument was derived from the resemblance to certain leaves, etc. The hand is considered to be the first, the best and the most important of all surgical instruments. As some of the modern physicians have attempted to show that some of them resemble modern obstetric instruments, a few remarks will be made. (1) Mandalagra—this is supposed to resemble a decapitator. It is described as a round or circular headed cutting instrument. Wise, in 1843, gave a diagram which is something like that given by Mukherji after Susruta. Another diagram appears in an English translation of Susruta by K. Bhisagratua in 1907 which has evidently been modified by Mukherji to resemble a decapitator. The description does not warrant such assumption. Apparently it is a sharp cutting instrument which may be used for perforation. Two other instruments, viz.—Bridhipatra and Mudrika, are described, which could be used for similar purposes. (2) Garbha-sauku is depicted by Mukherji to represent a blunt hook, but it is classified under sharp instruments. Binod Sen illustrates it as a sharp hook, which must be correct. (3) Jujna-sanku means paired sharp hooks, but it has been figured by Binod Sen to look like Palfyn's "iron hand." Sir Bhagbat Sing Jee, in his book on History of Aryan Medicine, hints that this indicates the use of forceps as a conservative instrument by the ancient Hindus. This claim has not been established. Perhaps modern Hindu writers have been greatly carried away by their admiration for ancient Hindu medicine and in their zeal imagined that such instruments represented modern perfected types.

Retained Placenta.—Gentle pressure on each side of the abdomen combined with *shaking by a strong person,* seizing her behind, is the treatment recommended. Comment is unnecessary.

Puerperal Diseases.—All diseases produced during the puerperal period are described under a common heading—Sutika voga, and are considered very serious, often incurable. The descriptions are meagre and the treatment recommended is unsatisfactory. Mastitis and mammary abscess received some consideration. (It may be stated here that in 1908 Dr. Pearse, then Medical Officer of Health of Calcutta, drew attention to the incidence of Sutika or chronic diarrhoea with fever, which caused a death rate of 1.3 per cent of the total number of registered births in Hindu and Mohammedan puerperal women in Calcutta. It should, however, be mentioned that there is no accurate method of registration of deaths, so that the cause of death after childbirth, is put down under a generic term "Sutika," which apparently covers a number of distinct diseases. In 1920, Dr. I. B. Basu of Calcutta made a careful study of fifteen cases of so-called "Sutika." He came to the conclusion that the symptoms pointed to the existence of anemia, combined with an extensive chronic enterocolitis caused by colon bacillus. The terminal infection is due to tubercle bacilli.

Management of the Infant.—Detailed directions are given with

regard to this subject. The selection of a wet nurse also receives careful attention.

Diseases of Infants.—Almost all diseases in infants are supposed to be produced by constipation. Purulent ophthalmia is mentioned and is believed to be caused by bad milk.

From the above brief survey of midwifery in ancient India, one may reasonably observe that Indian medicine was in possession of an imposing treasure of empirical knowledge and technical achievement. Naturally, the knowledge of anatomy, physiology and pathology was practically nil, but diagnosis was founded upon carefully collected evidence of the senses and anamnesis; while prognosis was most fully developed. In the treatment of disease, prevention played an important part. Hygiene and diet were considered as important as drugs and other therapeutic measures. The pharmacopia was a rich one, consisting chiefly of substances of vegetable origin, although no small number of animal and mineral substances were employed. Surgery constituted the summit of attainment of Indian medicine and Hindu practitioners were accustomed to perform difficult operations with boldness and skill.

MIDWIFERY IN INDIA DURING MOHAMMEDAN RULE (8TH TO 18TH CENTURY)

During the Mohammedan rule in India, surgery and obstetrics appear to have fallen to a very low ebb. Although it is certain that Arabian physicians were acquainted with old Hindu authors, the Mohammedan conquerors of India did not adopt any special measures for spreading medical science of the Arabian school amongst the conquered race and we may safely consider this period of 1000 years (8th to 18th century, A.D.) as a period of stagnation or rather decline. Arabian physicians left the care of obstetric cases to midwives.

(*To be concluded in February issue.*)

Selected Abstracts

Abdominal Cesarean Section

Gandy: The Administration of Anesthetics in Cesarean Section. Journal of Obstetrics and Gynaecology of the British Empire, 1922, xxviii, 534.

Stovaine or gas and oxygen with local analgesia are the preferable anesthetics for cesarean section in obstructed labor. The association of contracted pelvis and malformations of the thorax with diminished respiratory capacity, cardiac embarrassment or tuberculosis frequently makes ether an undesirable anesthetic. Gas and oxygen with local analgesia is the preferable anesthetic when cesarean section is done for severe antepartum hemorrhage. Ether or gas and oxygen with ethanesal are employed when the operation is done for eclampsia. Chloroform is contraindicated in the latter condition.

Gas and oxygen with ethanesal are eliminated from the fetal system faster than ether. Scopolamine and morphine affect the respiration of the child and should not be given within six hours of the time of delivery. Chloroform favors uterine bleeding and should never be used after opening the uterus. Occasional administration of morphine after removal of the fetus prevents postoperative nausea and vomiting. H. W. SHUTTER.

Holland, Eardley: Results of a Collective Investigation into Cesarean Sections Performed in Great Britain and Ireland from the Year 1911 to 1920 Inclusive. Journal of Obstetrics and Gynaecology of the British Empire, 1922, xxviii, 358.

Details are given covering 4197 cases of cesarean section. The cases are classified according to indications as follows: contracted pelvis (3372), eclampsia and other toxemias of pregnancy (227), antepartum hemorrhage (208) and conditions other than the above (366).

Three thousand three hundred and seventy-two cases of contracted pelvis were operated with a gross mortality of 4.1%. Twelve hundred and two cases operated before the onset of labor gave a gross mortality of 1.6%. Three hundred and eighty-nine cases sectioned early in labor had a maternal mortality of 1.8%. Cases operated late in labor gave a 10% mortality. A 14% mortality followed the operation in 35 cases where attempts at induction of labor preceded section. One hundred and one cases operated after unsuccessful attempts at delivery with forceps resulted in a maternal mortality of 25.7% and a fetal and infant mortality of 38%. Three mothers out of six died where cesarean section was done after attempted delivery by craniotomy.

The more common causes of death in 122 out of 139 fatal cases operated for contracted pelvis were general peritonitis 49, sepsis 16, septicemia 10, pulmonary embolism 8, pneumonia (all types) 17, intestinal obstruction 3, and ileus 2. Cesarean hysterectomy was done 46 times in the 3372 cases with a mortality of 17½%. Of the 3392 children including 19 cases of twins, 122 were born dead and 131 died in the first two weeks, a mortality of 3.9% and 4.2% respectively. The fetal and infant death rates were directly proportional to the duration of labor.

The maternal mortality in 195 cases of eclampsia treated by cesarean section was 32%. The mortality was three and one half times higher in the cases operated after the sixth convulsion. Of 37 deaths in this group the toxemia was blamed for 20, peritonitis 1, embolism 1, pneumonia (all types) 8, suppression of urine 3, etc. The fetal and infantile mortality was 50%. Prematurity was given as the chief cause of infant death.

One hundred and thirty-nine abdominal sections were done for placenta previa. A mortality of 11.5% for the group is divided as follows: complete or central placenta previa 14%, incomplete placenta previa 9.3%. The fetal and infant death rates totaled 27.3%. Prematurity is again the most common cause of infant death.

Cesarean section for accidental hemorrhage gave a maternal death rate of 27% in 66 cases. To control hemorrhage or remove uterine pathology cesarean hysterectomy was necessary in 30 cases with a resultant mortality of 46.6%. In all other cases the mortality was 11%. The fetal and infant deaths in these cases totaled 86%. Abdominal section with hysterectomy offers the easiest means of delivery in the badly shocked cases and provides a means of controlling hemorrhage from a uterus completely atonic. Operation is not done primarily to save fetal life.

Section was done 259 times for obstructed or prolonged labor. Obstruction was caused by fibroid tumors in 88 cases and ovarian cysts in 25. Carcinoma of the cervix indicated operation 25 times, cancer of the rectum, bladder and colon 9 times. The maternal mortality with cesarean section for fibroids was 10%, the fetal and infant mortality 25%. Of 25 cases of carcinoma of the cervix, 6 mothers and 14 babies died in the hospital. No deaths followed the operation for carcinoma of the colon, rectum and bladder. Twenty-five operations in this group followed ventral fixation, interposition or plastic repair. Impending rup-

ture of the uterus and rigidity of the cervix are each given three times as an indication for section. Congenital malformations obstructed labor eight times. Thirty-three cases came to operation because of malpresentation or excessive size of the fetus.

Grave maternal disease indicated operation 61 times. Of these, 40 were cardiacs in whom abdominal section resulted in a maternal mortality of 22.5% and 7 dead babies. Section was done twice for pulmonary tuberculosis and five times for intestinal obstruction. Operation was done ten times for habitual death of the fetus during labor. No mention is made of the number of cases in which a previous cesarean section indicated a repetition of the operation. The report is given in 89 pages of excellent detail invaluable to the obstetric surgeon. H. W. SHUTTER.

Bride: Cesarean Section in Manchester. Journal of Obstetrics and Gynaecology of the British Empire, 1922, xxviii, 463.

Six hundred and forty-eight cesarean sections were performed in Manchester during the years 1911 to 1920 inclusive. Five hundred and fifty-seven abdominal sections were performed for contracted pelvis with a mortality of 4.1%; 15 for eclampsia with mortality of 66.6%; 25 for antepartum hemorrhage with mortality of 28%, and 51 for other indications with 10 deaths, a mortality of 19.6%. There were in all 50 deaths in the 648 cases, a gross mortality of 7.7%.

Twelve of the 23 deaths in cases where the operation was done for contracted pelvis were due to general peritonitis. Nine of these 12 women had had repeated vaginal examinations with the membranes ruptured. In four of the cases forceps had been employed unsuccessfully at home. The author feels that cesarean section for contracted pelvis has little risk in cases not previously examined or where no attempt has been made at delivery from below. In spite of a maternal mortality of 66.6% in the cases sectioned for eclampsia the author apparently considers hysterotomy a favorable method of delivery in this condition. Cesarean section is recommended for placenta previa of the central type occurring in the primipara with a viable fetus.

Forty-nine per cent of 190 cases operated prior to 1918 have since become pregnant. Thirteen of these aborted from causes not necessarily intrinsic. Abortion does not follow cesarean section with marked frequency. Of the cases coming to term three delivered spontaneously and 68 were subsequently sectioned. Precaution against pregnancy seems to be the most likely factor in seeming sterility subsequent to cesarean section.

Of 4 women allowed to deliver spontaneously, one died from rupture of the uterus. In view of the favorable mortality of 2% in abdominal sections performed before the onset of labor one cannot justify a 25% mortality in women allowed to deliver spontaneously after section. H. W. SHUTTER.

Williams, J. Whitridge: A Critical Analysis of Twenty-One Years' Experience with Cesarean Section. Bulletin of the Johns Hopkins Hospital, 1921, xxxii, 173.

General conclusions are as follows: 183 cesarean sections were performed on 145 women in a series of approximately 20,000 deliveries and comprised 104 single and 79 repeated sections. There were 121 typical conservative sections, 4 extraperitoneal sections, 1 postmortem section, and 57 Porro sections. The gross mortality was 5.46%, but upon deducting cases in which death was not attributable to the operation, the net mortality was 3.45%; or 4.7% in the conservative and 1.82% in the Porro sections. All deaths except one from hemorrhage were

due to infection. Disproportion due to contracted pelvis was the indication for interference in nine-tenths of the black and six-tenths of the white patients. Several varieties of rachitic pelvis afforded the predominating indication in the blacks as compared with simple flat pelvis in the whites. The most frequent nonpelvic indications were eclampsia and serious cardiac decompensation. Cesarean section is not the ideal treatment for eclampsia and is indicated only in rare instances in which the cervix is rigid and undilated and venesection has not led to improvement. It is likewise rarely indicated in placenta previa. Only 7% of the children were deeply asphyxiated. The uterine cicatrix ruptured once in 48 women with repeated sections, as well as in 12 deliveries through the natural passages subsequent to section. Williams is convinced that cesarean section is abused throughout the country and that it is the cause of many unnecessary maternal deaths. C. O. MALAND.

Harris: A Study of the Results Obtained in Sixty-four Cesarean Sections Terminated by Supravaginal Hysterectomy. Bulletin of the Johns Hopkins Hospital, 1922, xxxiii, 318.

Supravaginal hysterectomy was employed more particularly in 2 classes of cases: first, to effect sterilization after repeated cesarean sections; and, secondly, to obtain more ideal results upon patients who were already infected or who were admitted to the service after being long in labor. In 223 cesarean sections —supravaginal hysterectomy was done in 64 instances with 3 deaths or a gross mortality of 4.68%. Only one (operative accident) was directly associated with the operation,—a cardiac condition and a pre-existing renal condition respectively accounting for the other two deaths. Thus only 1.56% resulted from the operation. Hysterectomy was done on 23 patients to effect sterilization; on 18 patients already infected or late in labor; 5 cases of atresia of cervix; 4 neglected transverse presentations; 2 hour glass contractions of uterus; 2 failure of bag or bougie to induce labor; 1 dystocia following ventral fixation of uterus; 9 for some other cause. C. O. MALAND.

Brouha: Must Abdominal Cesarean Section Be Reserved for the Clean Case? Gynécologie et Obstétrique, 1920, ii, 385.

Unfortunately, in many instances, dystocia makes itself evident only after labor is well along and after the bag of waters has been ruptured for various lengths of time. Should we say in such cases that we should not perform an abdominal cesarean? P. Bar does not hesitate to answer in an affirmative and his opinion is so outstanding and based upon so long and comprehensive an experience that it is vested with great authority. However, the author wishes to give the results of sixty conservative cesareans that make him hesitate to agree absolutely with this opinion.

In twelve of these cases all the ideal conditions enumerated by Bar were existing. In forty-eight of them, however, labor had been in progress for variable lengths of time, the greater number had been subjected to repeated examinations at home. In two-thirds of the cases the membranes had ruptured from two hours to five days before; dilation of the cervix varied from the size of a dime to nearly complete dilation. These cases were clearly not strictly clean and yet in the absence of fever and of all signs of uterine infection we did not hesitate to perform a cesarean to save the child. Success has followed our temerity.

Two of the twelve strictly clean patients died. None died among the questionable cases.

These results lead the author to believe that if one has full charge of the case from the beginning, one could allow the labor to progress a long time under strict aseptic conditions before the case would become sufficiently contaminated to lead one to decide against an abdominal cesarean in the face of a definite indication. R. T. LAVAKE.

Bell, Blair: The Sterilization of Infection Incurred Before Cesarean Section Is Performed. Journal of Obstetrics and Gynecology of the British Empire, 1922, xxviii, 528.

The author in doing cesarean section on presumably infected cases employs the following method of wound sterilization. After delivery of the placenta a gauze saturated with sodium hypochlorite solution (Milton's fluid) is applied to the edges of the wound for one or two minutes. The gauze is then placed in the uterine cavity and before closing is pushed out into the vagina with a uterine dressing forceps. The pack is not removed from the vagina until after the patient is returned to her bed. To date the method has been employed in ten cases with good results. H. W. SHUTTER.

Bauch: Cesarean Section upon the Dead and Dying Patient in Eclampsia. Zeitschrift für Geburtshülfe und Gynaekologie, 1922, lxxxiv, 596.

The author gives a brief historical review of postmortem cesarean section. He finds that it is one of the oldest recorded obstetrical operations. Although absolutely trustworthy statistics are not available, it may be assumed that the operation must have been attended by a certain percentage of successful results, even in early times or it should long since have been abandoned. The statistics of Schwarz for the years of 1836 to 1848 showed 107 operations without a single living child, and in Dohrn's later series of 90 cases, all the children were also dead. The more recent literature has given a number of successful results, yet Cathalos' series with 61% of living children and Böhmer's with 68% are probably explained by the greater tendency to publish successful cases.

Recent animal experiments have shown us more about the circumstances in which the fetus survives the dead mother for a time. When death occurred suddenly, as from hemorrhage or chloroform intoxication, the fetus survived longer, usually about five minutes. When the death agony was long drawn out, or there was a high fever, the fetus survived only a short time or even died before the mother.

Operation upon the moribund patient has been proposed and occasionally carried out to avoid the danger to the child of the death-agony. It involves far more complicated questions than does postmortem cesarean, and may assume a medicolegal aspect, for should the patient survive the operation for some hours, the operation may be charged as the determining cause of the fatality. Though in consideration of the rights of the child, postmortem cesarean may and should be performed even without definite consent when this cannot be obtained in cases of sudden death, a section upon the moribund patient should never be performed without consent of the relatives and consultation with another physician to confirm the dying condition of the patient. Whereas, in postmortem cesarean, haste is the great essential, section on the moribund patient should not be performed without careful surgical technic. The placenta should always be removed, as otherwise it may be expelled from the vagina after the death of the mother. Determination that the fetus is alive should be made before section on the moribund patient, yet is not an essential prerequisite for post-

mortem cesarean, for extraneous circumstances may make it very difficult to hear the fetal heart beat in a restless dying patient.

Cesarean section upon the dead or dying patient has been performed in the following diseases: heart failure, pneumonia, chronic diseases of heart; kidney or lung diseases; embolism, rupture of large vessels, cerebral tumor, meningitis, eclampsia, chorea gravis, peritonitis, pernicious anemia and burns. The eclamptic patient may die undelivered either suddenly in the first convulsion, or after an eclamptic coma of some duration. There are a number of factors which unfavorably influence the prognosis for the child in eclampsia: the frequent prematurity, the toxicity, and the asphyxia attending the convulsions. Yet in 13 postmortem sections and one upon a dying patient in 500 cases of eclampsia at the Kühn clinic, there were obtained three living children, or 21.4%. Since one of these 14 cases was a hydatid mole and another a macerated child, the three living babies represent 25% of the total possibilities. One of the three babies was, however, deeply asphyxiated and died within a few hours. Another lived ten months and died suddenly of "inward" convulsions. The final fate of the other is not recorded. But the saving of even an occasional life surely justifies the operation. MARGARET SCHULZE.

Kerr, Munro: Indications for Cesarean Section. Journal of Obstetrics and Gynaecology of the British Empire, 1922, xxviii, 338.

With gradually improving results from cesarean section the upper limits of pelvic deformity for which the operation is indicated have been raised to 8.1 cm. Had we means of insuring a firm uterine scar many cases now handled by the older methods would be better treated by cesarean. Induction of labor does not come into competition as its results are seldom satisfactory when the conjugata vera is less than 3¼ inches. Induction of labor is indicated when the accoucheur has experienced difficulty in delivering by forceps at a previous labor and feels that a slightly smaller child could be delivered easily. With few exceptions when delivery from below is impossible, cesarean section should be done if the child is alive, craniotomy if it is dead. Pubiotomy, seldom suitable for contradictions of the inlet, is not employed enough for outlet contractions.

Cesarean section is indicated for obstructing fibroid tumors. Too many abdominal deliveries are done for abdominal cystomata. Frequently these tumors, even with the patient in labor, can be pushed out of the pelvis with the aid of Trendelenburg posture and deep anesthesia.

The only cases of eclampsia suitable for cesarean section are those with well-advanced pregnancy, in the absence of cervical dilation or previous attempts at delivery and in which there has been no improvement after six hours of blood letting, morphine and intravenous saline.

Marginal and lateral placenta previa are well handled by the conservative methods of treatment. Cases treated by the older methods carry a maternal mortality of from 4% to 10% and a fetal mortality of 40%. One hundred and thirty-one cases of placenta previa treated by cesarean section gave a maternal mortality of 10%, a fetal mortality of 15%. In the author's opinion cesarean section is the best method of treatment for most cases of central placenta previa.

Excluding the cases of accidental hemorrhage which can be controlled by conservative treatment, cesarean section and hysterectomy alone remain suitable procedures. In these cases abdominal section offers immediate delivery, control of hemorrhage and is frequently the means of saving maternal life.

Under special circumstances, brow, face and occipito posterior presentation,

rigid cervix, high retraction ring and scars from previous pelvic operations become indications for the operative procedure. Cesarean section is indicated when dystocia from ventral fixation or interposition operations prevent delivery. Cardiac disease and other maternal emergencies occasionally indicate operation. Such conditions as hydramnios, hydrocephalus, varicose veins and impacted shoulder presentations seldom justify abdominal delivery. Improved operative methods and results are gradually extending the indications for cesarean section.

H. W. SHUTTER.

Taylor, Charles J. G.: Cesarean Section: With Special Reference to Present-day Indications for Operation. The British Medical Journal, June 10, 1922, No. 3206, p. 909.

The author considers absolute indications such as (1) pelvic deformity where the conjugate is perhaps 2¼ inches or less; (2) hard solid tumors; as relative indications: (1) contracted pelvis with a conjugate between 3¼ and 3½ inches; (2) tumors such as fibromyomata and ovarian cysts; (3) placenta previa (he believes this operation should be performed in cases of central and lateral placenta previa, but not in the marginal form); (4) eclampsia (he thinks in some of these cases cesarean section is a matter of choice); (5) concealed accidental hemorrhage (section associated with hysterectomy); (6) ventrofixation. (cesarean section may be necessary where the cervix is tilted backward); (7) prolapse of cord (he does not recommend this as a routine practice for this condition); (8) impacted shoulders (he advises against general use of cesarean section in these cases, mortality of both mother and child being about 50%); (9) abnormal conditions of the child, such as postmaturity, rarely brow presentation, persistent occipitoposterior position, impacted breech (all of these conditions should be regarded as exceptional indications); (10) retraction and contraction rings (a conceivable indication for section); (11) scar tissue and a rigid cervix; (12) serious diseases of the mother, such as cardiac disease, advanced pulmonary tuberculosis. He mentions postmortem cesarean section as a justifiable procedure. Other occasional causes are mentioned such as extreme edema of the vulva, also large varix of the vagina and vulva. He raises a question as to whether cesarean section should be performed before labor has started and sees no contraindications to the performance of section before the onset of labor.

He thinks there is no reason for routine sterilization of the patient.

There are cases on record of repeated cesarean sections four, five, and even more times. The possibility of rupture of the uterine scar during pregnancy should be borne in mind. The writer seems to accept the dictum "once a cesarean, always a cesarean" only for pelvic contraction. In conclusion the author emphasizes the supreme importance of a careful watching of all cases during pregnancy, and stresses the urgent need for maternity beds in large towns and counntry districts.

F. L. ADAIR.

Henkel, M.: Cesarean Section and Its Indications In Modern Obstetrics. Medizinische Klinik, 1922, xviii, 917.

In addition to absolute and relative indications we now have prophylactic indications as well. Practically there are three forms of cesarean section: the classic, the intraperitoneal cervical and the extraperitoneal cervical.

It is easy to deal with the absolute indications for the operation but not so with the relative ones. The experienced obstetrician in dealing with contracted pelves prefers first to see the effect of labor pains before making any decision. The question of cesarean section is further complicated by the fear of infection.

There is no doubt that in the presence of infection the prognosis is much worse; but at present there is no means of recognizing the presence of infection early. The patient may be severely infected and yet show no symptoms of this and on the other hand, she may have no infection and yet show streptococci in the uterine secretion and in the blood and she may have fever and tachycardia.

Henkel believes the intraperitoneal cervical operation is the one of choice and describes his method of performing it. The patient is kept in the horizontal position throughout the operation. The indications for cesarean section may be extended because a difficult delivery *per vias naturales* under certain conditions may be more dangerous to the mother than a cesarean section. In placenta previa, e.g., the abdominal operation proves blood-saving; but it should be reserved for placenta previa centralis in primiparas. In eclampsia where the cervix is undilated and the head high, cesarean section is the quickest and most certain method of delivery for both mother and child. The author emphasizes that the technic of the operation is now standardized but the difficulties arise in placing the indications. J. P. GREENHILL.

Jones, Arnold: A Plea for the More Frequent Use of Cesarean Section. British Medical Journal, July 16, 1921, No. 3159, p. 75.

The author advocates more frequent use of cesarean section in the treatment of placenta previa and eclampsia. He describes an operation which he thinks makes the uterine scar capable of standing the strain of future labors. He uses a midline incision, incising the external layer of the uterine muscle transversely just below the center of the uterine body. He makes a V-shaped incision about one-eighth inch deep. This layer is peeled upwards toward the fundus. The middle and inner layers are now incised longitudinally. The longitudinal incision is closed with a continuous catgut suture, the transverse incision in practically the same way. There is very little trouble with blood loss in this operation. He has performed the operation in eight cases; four of eclampsia, two of placenta previa, and two of contracted pelvis. None of these cases have had subsequent labors. F. L. ADAIR.

v. Jaschke, R. T.: The Justification of Abdominal Cesarean Section in the Treatment of Placenta Previa. Monatsschrift für Geburtshülfe und Gynäkologie, 1922, lviii, 249.

Cesarean section nowadays is for the large proportion of obstetricians, the method of choice in dealing with severe cases of placenta previa especially placenta previa totalis. Hitschmann, who does not favor cesarean section as the usual method of dealing with placenta previa had a maternal mortality of 5% in his own cases of placenta previa treated by cesarean section. But he performed the classic operation. In the cases in which the low cervical cesarean is performed the mortality is not above 2% and it does not matter whether the operation is intra- or extraperitoneal. As Hitschmann himself mentions, the general maternal mortality for cesarean section performed for placenta previa is 3.6% whereas for the older methods (Braxton-Hicks version, metreurysis, etc.), it still is 7.6%.

Hitschmann's point of view as regards treatment is untenable because he fails to consider the life of the child. The lowest fetal mortality obtained with metreurysis is about 20% while the general fetal mortality where the older methods are used, is 30 to 40%. It is v. Jaschke's opinion that the child's life should be neglected only when a cesarean section would endanger the life of the mother.